A TEST OF THE MATERNAL STRESS HYPOTHESIS FOR

HUMAN MALE HOMOSEXUALITY

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Recent evidence implicates a biological, and particularly, a neurohormonal role for the etiology of human male homosexuality. In contrast to most other traits, however, there is a priori reason to doubt high heritability of differences in sexual orientation, due to the presumed selective disadvantage of homosexuality. The discovery that prenatal stress behaviorally feminizes male offspring in rats has been of great interest as a plausible environmental model which could account for the neural feminization required by the neurohormonal theory of male homosexuality. Dorner has presented evidence suggesting a strong maternal stress effect for human male homosexuality; however, his methodology was grievously flawed.

The maternal stress hypothesis was tested using mothers' retrospective reports of events during pregnancy. Such reports were obtained from 83 mothers of nonheterosexual males (Kinsey Fantasy Scores ≥ 3; 72 of these subjects were Kinsey 5s or 6s.), 60 mothers of heterosexual males (Kinsey Fantasy scores < 3), 53 mothers of heterosexual females, and 19 mothers of female nonheterosexuals. A within-family analysis was also done, as mothers also rated stress for their pregnancies with heterosexual siblings of subjects. Results of both between-family and within-family analyses were strikingly negative for males. Unexpectedly, however, there was a significant correlation between sexual orientation and prenatal stress for females, with mothers of nonheterosexual females reporting greater stress.

An additional analysis of maternal stress-proneness provided some support for a modified maternal stress hypothesis: Stress-proneness in mothers (measured by personality scales) correlated positively and significantly with childhood effeminacy of male offspring. This correlation was negligible for females.

While not disproving the maternal stress hypothesis, results of this study are not consistent with a strong effect of maternal stress on male sexual orientation. Because male homosexuality is strongly familial (confirmed in the present study), it is recommended that genetic explanations should be pursued more vigorously.

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Chapter 1

Introduction and Literature Review

Homosexuality refers to consistent erotic arousal by members of one's own sex. It contrasts with heterosexuality, in which erotic orientation is to members of the opposite sex. Bisexuality refers to a range of intermediate preferences. It is important to note that sexual orientation is defined psychologically rather than by actual sexual behavior. Although the behavioral and psychological dimensions are strongly correlated, they are distinct. A person might be homosexually aroused yet never have homosexual relations. Only those who are consistently aroused by members of the same sex are here considered homosexuals, regardless of their behavioral pattern.

The role of biological mechanisms in the etiology of male homosexuality has become a topic of great interest to behavioral scientists (Ellis & Ames, 1987; Money, 1987). This is due in part to the failure to confirm hypotheses of psychosocial causation (Bell, Weinberg, & Hammersmith, 1981). But it is also attributable to several findings in both human and animal research which suggest neuroendocrine involvement in the development of sexual orientation.

In humans the most suggestive evidence has been provided by the so-called hermaphroditic or intersexual syndromes (also referred to as sexual inversions) in which prenatal endocrine anomalies can affect subsequent psychosexual development. For instance, adrenogenital

syndrome exposes a female fetus to abnormally high levels of androgens, and is associated with a marked increase in bisexuality and homosexuality in adolescence and adulthood (Money, Schwartz, & Lewis, 1984). There is no analogous syndrome predictive of homosexuality in males; hence, the relevance of human intersexual syndromes to the phenomenon of male homosexuality is only indirect. In animals, male homosexual behavior has been induced via several experimental paradigms which generally involve manipulation of prenatal hormones. Such manipulations (e.g., castration) cannot be considered models for the etiology of naturally-occurring homosexuality.

However, one paradigm in the animal literature has been considered a promising etiological model for human male homosexuality: maternal exposure to stress during pregnancy (Dorner et al., 1980). Experimentally manipulating maternal stress has been remarkably successful in inducing homosexual-like behavior in a large proportion of male rats (e.g., Ward, 1972). Because there are wide differences among humans in exposure to stress, this paradigm remains a possible model for human male homosexuality.

I begin by examining the theory that human male homosexuality results from partial feminization or incomplete masculinization of the brain. After briefly reviewing the role of hormones in psychosexual development, I examine the phenomenon of maternal stress-induced homosexuality in animals and the evidence that such a mechanism also occurs in human homosexuality. I then investigate the role of maternal stress in human homosexuality. Because maternal stress appears to be

relevant only for male and not for female homosexuality (Politch & Herrenkohl, 1984), I focus almost exclusively on the former.

Homosexuality and Behavioral Effeminacy

In this and the following section I consider the hypothesis that male homosexuals differ from male heterosexuals primarily on the dimension of "masculinity-femininity," and that this difference has neural underpinnings. Specifically, male homosexuality is hypothesized to be caused by incomplete masculinization of the brain during sexual differentiation. Henceforth, I refer to this hypothesis as the feminization theory of homosexuality (for short, feminization theory). Strictly speaking, feminization theory is the conjunction of two distinct theories: one theory that phenotypically, male homosexuals differ from male heterosexuals in the feminine direction, and another theory that the feminization is caused by (probably prenatal) hormonal effects on the brain. The first theory is descriptive, while the second is etiological.

Support for feminization theory includes evidence of a psychological, a behavioral, and a biochemical nature. Psychological and behavioral evidence are more relevant to the descriptive component of feminization theory. Biochemical evidence is more informative regarding the etiological component. In this section I explore the psychological and behavioral evidence for feminization theory. In the next section I consider biochemical and other physiological evidence.

In discussing homosexuals and heterosexuals it will often be

necessary to employ the terms 'masculine' and 'feminine'--whether one's focus is at the behavioral or neurological level. Thus, a brief clarification of their meaning is in order. 'Masculine' traits are traits which have relatively large sex differences, in favor of males. These traits are distinguished from 'feminine' traits, in which the sex differences are in the opposite direction. These terms are often used to denote the opposite ends of a single continuum, and to a large extent masculine characteristics are negatively correlated with feminine ones, even within the sexes. However, the correlation is not perfect. As a result, in a given domain it is possible for an individual to have both masculine and feminine traits, to have one but not the other, or to have neither (Ellis & Ames, 1987). An individual is effeminate to the extent that he or she exhibits feminine traits.

The sexual object choice of male homosexuals is identical to the object choice of female heterosexuals. This is the most obvious fact about male homosexuals which suggests that they may have been feminized during development. Sexual orientation is but one of several dimensions in the realm of masculinity and femininity (Finn, in preparation). Are male homosexuals effeminate in other important ways?

Adult homosexuals. Relative effeminacy of adult male homosexuals is found in global measures of overt behavior. In an ambitious study containing almost 700 male homosexuals (Bell, Weinberg, & Hammersmith, 1981), interviewers were asked to rate respondents' effeminacy according to their appearance and mannerisms. Over 30% of homosexuals were rated as being "somewhat" or "very" effeminate, while

only 1% of heterosexuals were so rated. Similarly, 65% of heterosexuals were perceived as "somewhat" or "very" masculine, compared to only 25% of homosexuals. These percentages translate into about a one standard deviation difference in effeminacy, indicating substantial overlap. Indeed, 48% of homosexuals and 34% of heterosexuals were rated as being neither especially feminine nor especially masculine.

On some psychological tests which show appreciable sex differences, adult male homosexuals perform intermediate to heterosexual males and females. For instance, Spence and Helmreich (1978) found gay men to be significantly lower in Dominance-Poise and higher in Nurturance-Warmth (as measured with the Personal Attributes Questionnaire) than a control group of heterosexual men, a pattern which also obtains in women. Two other studies generated results in the same direction, although they failed to reach significance. These failures to reach significance are attributable to the fact that such differences between heterosexuals and homosexuals are not large.

Homosexuals also appear to have a feminine pattern on some tests which measure aspects of cognitive functioning. For instance, Sanders and Ross-Field (1986) demonstrated that heterosexual males outperformed both homosexual males and heterosexual females on a task designed to measure visuo-spatial ability. Homosexuals also have the feminine pattern of verbal-performance IQ difference, with verbal IQ being a relative strength (Willmott and Brierley, 1984), although there was again considerable overlap. Thus, adult male homosexuals appear to be relatively effeminate, not only when effeminacy is assessed directly (as

in the behavioral rating scales), but also when relevant traits are only moderately correlated with gender (such as cognitive ability patterns).

Homosexuals as children. It is instructive to examine the extent to which the apparent effeminacy in male homosexuals is analogous to effeminacy in females. If effeminacy in homosexual males is analogous to that in females--i.e., if both phenomena arise from similar mechanisms--they should have similar developmental patterns. Thus, it is important to look at the childhood behaviors of adult homosexuals.

There have been several studies relating male homosexuality to childhood effeminacy, and the picture is clear: Childhood effeminacy is the single best predictor of adult homosexuality. For instance, Bell et al. (1981) included retrospectively assessed childhood gender nonconformity (essentially effeminacy) in their aforementioned study on the development of sexual orientation. They found it to be the most important developmental variable in their path model for predicting adult homosexuality. Similarly, Whitam (1980) asked adult homosexual and heterosexual males six questions regarding childhood effeminacy (e.g., "Were you interested in dolls?"). Most of the homosexuals (85%) endorsed two or more of the items, but few of the heterosexuals (6%) did. The lowest estimate in the literature for childhood effeminacy among homosexuals is 67% from Saghir and Robin (1973); thus, a majority of gay adult males appear to have been effeminate as children. Furthermore, Harry (1983) has reported that the items used to detect effeminacy are much less intercorrelated and longitudinally stable among heterosexuals than among homosexuals. Thus, the items do not appear to measure a

reliable and stable attribute of heterosexuals, as they do in homosexuals.

A study by Grellert et al. (1982) is informative regarding the relationship between effeminacy in heterosexual females and in homosexual males. These investigators examined various dimensions of childhood play for both sex differences and sexual orientation differences. They found that the two scales which best discriminated between the two sexes, Feminine Play and (masculine) Sports, were also the best discriminators for sexual orientation. In a similar study Blanchard et al. (1983) studied childhood patterns of aggression in both heterosexual and homosexual males. They found childhood physical aggressiveness to be significantly lower in the homosexual than in the heterosexual group. Females are also relatively unaggressive compared to male heterosexuals (Maccoby & Jacklin, 1974).

Thus, the prehomosexual boy tends to enjoy activities which are traditionally seen as feminine, such as playing with girls and wearing dresses. He tends to avoid traditional masculine activities such as rough-and-tumble sports. He has interests which would be quite unremarkable in a female child, yet he is likely to be called a "sissy" and ostracized by peers.

The negative consequences of effeminacy shed light on a curious observation, viz., that differences in effeminacy between heterosexuals and homosexuals appear to be more striking in childhood than in adulthood. The majority of adult gay men are not markedly effeminate, but the large majority of male homosexuals report having been effeminate as children. Whitam (1977) has hypothesized that there must be a

defeminization process prior to adulthood. Defeminization presumably represents a response to parental and peer pressure to conform to standards of gender-appropriate behaviors and interests. Harry (1983) found that gay males indeed report becoming less effeminate with age.

The studies of childhood effeminacy discussed heretofore have all used retrospective accounts of adults. Ross (1980) has argued that such accounts may be biased by falsely remembering effeminacy in order to confirm the experimenter's hypothesis. It is also possible that a tendency may exist for adult heterosexuals to forget, or not to report, childhood effeminacy. Prospective studies of effeminate boys vitiate this possibility. Green (1985) followed up 44 boys who had been selected because of extensive cross-gender behavior. Boys were identified as early as age 4 (average age: 7.5 years). At follow-up (average age: 18.5 years), 68% had Kinsey fantasy scores which indicated either bisexuality or homosexuality. None of the 34 boys in a control group had scores in this range. Furthermore, among those subjects with a history of cross-gender behavior, the average age of those denying homosexual or bisexual orientation (15.8) is significantly lower at follow-up than the age of those admitting such an orientation (19.4). This suggests that some of the remaining "heterosexuals" in the effeminate group will eventually shift their orientation to the bisexual or homosexual group.

Green's results replicate those of Money & Russo (1979) and Zuger (1984), both of whom found increased homosexuality among males with a childhood history of extremely effeminate behavior. It is likely that the degree of effeminacy seen in such males is rare--rarer, for instance,

than the 4% estimated incidence of exclusive or predominant homosexuality. If so, Green's study may overestimate the incidence of future homosexuality in boys with cross-gender behavior of a degree more typical of prehomosexuals. Nevertheless, prospective studies are consistent with retrospective ones in confirming that childhood effeminacy is an extremely important factor in the development of homosexuality.

The question posed near the beginning of this section, whether homosexuals are effeminate in ways other than sexual orientation, can now be answered affirmatively. On average, adult homosexuals are relatively effeminate in their appearance and mannerisms. Their scores on several psychological and cognitive tests are intermediate to heterosexual females and males. More impressively, most gay men begin as effeminate boys, and a large percentage (perhaps a majority) of effeminate boys become homosexual adults. Phenomenologically and developmentally, there are similarities in the effeminacy of prehomosexuals and females. This suggests that the two phenomena may share a common etiology. Below, I explore what this etiology might be.

Evidence for a Biological Role in Homosexuality

I have argued that homosexuality involves behavioral feminization, and that such feminization is likely to be etiologically similar to that which occurs in heterosexual females. The causes of behavioral effeminacy in heterosexual females are controversial, and may depend on the specific trait of interest. There are, however, only two broad

etiological candidates mentioned: innate, biological factors generally related to brain function, and psychosocial mechanisms such as socialization and imitation.

There are rather strong a priori grounds for suspecting that in homosexuality, biological factors will play the greater role. This is because the most familiar psychosocial mechanisms cannot plausibly be operating. Male prehomosexuals are effeminate despite being socialized to the contrary, despite the masculinity of their male role models, and despite the punishment which often follows effeminate behavior in males. Although there have been some psychological theories which circumvent these problems by emphasizing subtle aspects of parenting (e.g., Lidz, 1968), these theories have generated remarkably little empirical support (Bell, Weinberg, & Hammersmith, 1981; Sieglman, 1981). Moreover, insofar as such theories have garnered support, the direction of causation is ambiguous. For instance, consistent with Freudian theory, male homosexuals do appear to have poorer childhood relationships with their fathers than do male heterosexuals (Siegelman, 1974). However, as Bell et al. have pointed out, it is possible that the fathers are reacting to the effeminate childhood behaviors of the prehomosexuals.

The etiological part of feminization theory--viz., that homosexuality involves feminization of the brain--cannot merely rest on the implausibility or lack of evidence of its psychosocial competitors. Fortunately for the theory, there is mounting evidence in both the human and animal literature consistent with neuroendocrinological underpinnings to homosexuality. Before considering this evidence, however, it

is useful to review briefly the biology of sexual differentiation. By focusing on the heterosexual from conception, we can be more informed in assessing possible causes of effeminacy in male homosexuals.

Sexual differentiation. There are three basic levels of sexual differentiation: the differentiation of internal sex structures, of external genitalia, and of brain. Each process is extremely complex; however, there is a common thread among the three. In order for development to proceed in a masculine direction, sufficiently high levels of testosterone are required. Otherwise, feminine development occurs.

In the eighth week after conception the male embryo begins to secrete two substances necessary for male differentiation: Mullerian inhibiting substance and testosterone. The former causes the Mullerian ducts to shrink instead of forming the female internal sex organs. The latter stimulates the development of the male internal sex organs such as the epididymis, vas deferens, seminal vesicles, and ejaculatory ducts. In addition, testosterone is converted into another androgen, dihydrotestosterone, which stimulates development of the penis, scrotum, and prostrate gland. Without testosterone the uterus, Fallopian tubes, and inner third of the vagina develop, as well as the external female genitalia. Thus, in contrast to male development, female sexual differentiation requires no hormonal activation.

More important for the topic at hand is the differentiation of the brain. Hormones affect the development of the brain and the pituitary gland, causing sex differences in certain brain structures and functions. The best documented difference is in the number and location of

synapses in the hypothalamus, such as the preoptic anterior nucleus (Goldman, 1978). Consistent with the other areas of sexual differentiation, a masculine pattern requires testosterone; without it, the brain develops in a feminine pattern (McEwen, 1981). The prenatal hormonal organization sets the stage for hypothalamic and pituitary brain function during and after puberty. Hence, females have cyclical sex hormone production, while males' production is relatively constant.

The extent of the behavioral consequences of structural differences between male and female brains is a controversial issue (Ellis, 1986). Prenatally determined structural differences may pave the way for later differences in sexual behavior and aggressiveness (Rubin, Reinisch, and Haskett, 1981). It is also reasonable to hypothesize that these neural differences may underlie differences between the sexes in typical sexual object choice. The brain structures which are believed to be involved in sexual orientation appear to differentiate around the second trimester of pregnancy (Dorner, 1976), implying that the second trimester of fetal development may be critical to eventual sexual orientation (Ellis & Ames, 1987).

Human evidence for biological etiology of homosexuality. Two bodies of human evidence support a hypothesis of neuroendocrine involvement in male homosexuality: The first includes certain sexual inversions of known hormonal causation, which are indirectly supportive of feminization theory. The second includes recent findings in homosexual subjects, which are directly supportive.

Adrenogenital syndrome, mentioned briefly above, is probably the

most interesting sexual inversion with respect to male homosexuality. Due to a genetic error of cortisol synthesis, large amounts of androgens are secreted from the adrenal glands of the affected. If this error occurs in a genetic female, the amount of testosterone secreted prenatally is comparable to that found in normal male fetuses. As a result, her external genitalia may be masculinized to a large degree. (The internal genitalia develop sufficiently early to escape masculinization.)

Since 1950, it has been possible to provide adrenogenital females with a treatment beginning soon after birth: Their genitalia are surgically feminized (e.g., "penises" are removed), and they are given antimasculinizing hormonal treatment for the rest of their lives. As children, these women are more likely to prefer competitive sports, and are less likely to prefer playing with dolls and dressing in women's clothing (Money & Ehrhardt, 1972). Money, Schwartz, and Lewis (1984) have followed up 30 cases of treated adrenogenital syndrome into early adulthood. Of the women who would admit their sexual orientation, 48% are either bisexual or homosexual, an enormous increase compared to controls.

How is adrenogenital syndrome, which affects females, relevant to the feminization theory of male homosexuality? The most obvious way is as an example of prenatal hormones causing an alteration in sexual orientation. Of course, one is not entitled to assume that female and male homosexuality have identical causes; nevertheless, the example is suggestive. A stronger sense in which the syndrome is relevant arises from the similarity between the adrenogenital female's sexual develop-

ment and that of the heterosexual male. The adrenogenital female, like the heterosexual male, is exposed to high levels of prenatal androgens. Like the typical male, she is more masculine and is much more likely to prefer women as sex partners than are normal females. This shift in sexual preference must be due to the androgenizing effects of the hormones. This suggests the possibility that male homosexuals fail to develop a preference for female sex partners because of a lack of prenatal testosterone. This possibility is also consistent with the effeminacy of gay males. The hypothesis that male homosexuality is caused by a lack of prenatal testosterone is elaborated more fully in a later section.

Do any findings of studies using male homosexuals as subjects support a hypothesis of biological, particularly neuroendocrine, influence? The possibility that male homosexuality is a neuroendocrine phenomenon has inspired two research paradigms using male homosexual subjects. The simpler paradigm involves looking for differences in blood testosterone-level between homosexuals and heterosexuals (e.g., Birk, Williams, & Chasin, 1973). There is a trend for homosexuals to have slightly lower levels than heterosexuals; however, the former are well within the typical male range, and well above the typical female range. It would appear then that level of circulating testosterone is not a salient factor in the genesis of homosexuality. This fact need not be interpreted as unfavorable to a hypothesis of neuroendocrine involvement (Ellis & Ames, 1987). The level of circulating testosterone is primarily due to the ability of the gonads to produce testosterone, which is ultimately due to the success of gonadal differentiation. But there is reason

to believe that homosexuality has little to do with incomplete gonadal differentiation. If it did, for instance, we would expect to see gross morphological differences in muscularity and body hair between heterosexuals and homosexuals. No such differences are evident. Moreover, the unique characteristics of homosexuals--their behavioral effeminacy and sexual orientation--implicate feminization of the brain, not the gonads.

A more complex paradigm originated by Dorner et al. (1975) has led to more promising results. These investigators studied the level of luteinizing hormone (LH) after an injection of estradiol in homosexual males and heterosexual males and females. The typical response of females to such an injection is a sharp rise in LH level over about four days. This response mimics the natural surge of LH after release of estradiol which occurs during the female's monthly menstrual cycle. In contrast, heterosexual males generally fail to experience a surge of LH after estradiol injection. Interestingly, about one-half of a group of male homosexuals studied by Dorner et al. responded similarly to (though less intensely than) heterosexual females. This result has been replicated by Gladue et al. (1984). Although there is no causal theory linking this LH surge to male homosexual behavior, these findings are important because they suggest that male heterosexuals' and homosexuals' neuroendocrine systems indeed differ. Moreover, the finding implicates the hypothalamus, since the hypothalamus triggers the pituitary gland to produce LH. In contrast to blood-testosterone level, which is determined by gonadal functioning, the LH surge is a neuroendocrinological phenomenon. That is, whether or not the LH surge occurs depends on

whether the brain is organized in a masculine or feminine way.

One-half the homosexuals in the aforementioned two studies failed to exhibit the LH surge. How does one account for this? One obvious possibility is that homosexuality is a heterogeneous category--there are different types of homosexuality, each with a different cause. However, this explanation is not necessarily required. Another possibility is that a homosexual orientation requires feminization of a certain part of the brain (Area A), and that other areas (affecting different functions) are often feminized simultaneously. If this were the case, one would expect all homosexuals to have a feminized Area A, but individual homosexuals could differ as to which other areas were feminized. Different patterns of feminization might occur depending on the timing and extent of feminization. In order to explore this possibility, it will first be necessary to discover specific neuroendocrinological differences in male homosexuals. The best place to look for such differences, of course, is in those phenomena which show substantial sex differences.

The possibility that different feminine behaviors are caused by areas of the brain which differ in their developmental timing might help to explain why, despite some effeminate behaviors, male homosexuals exhibit the desire for sexual variety, which is more typical of male than of female heterosexuals (Symons, 1979). If this sex difference is due to brain differences, then we would expect it to occur at a distinctly different time during development than the brain differences which determine sexual orientation.

Green (1987) relates a fascinating case relevant to the question of heterogeneity. In the aforementioned longitudinal study of extremely effeminate boys, one proband is an identical twin. His cotwin was discordant for childhood effeminacy. At last follow-up, however, the proband was determined to be bisexual, as was the masculine cotwin. Parsimony dictates the assumption that the cotwins' bisexual orientations have similar causes--that the cotwins are from a homogeneous class. (One plausible objection is the that the masculine twin may have been influenced by his effeminate cotwin.) Yet the behavioral antecedents and correlates differ. One might speculate that the effeminate twin's brain was feminized to a greater degree than the masculine cotwin's. This example suggests that phenomenological heterogeneity need not imply etiological heterogeneity.

The vehicle problem. Given the plausibility of a biological explanation of human male homosexuality, there remains the problem henceforth referred to as the "vehicle problem:" If male homosexuality is caused by a dearth of testosterone at a critical period of development, what vehicle causes this dearth of testosterone to occur?

In psychopathology and personality, there is a ready candidate to account for biological mechanisms: genetic variation. Both extraversion and schizophrenia are presumed to be strongly influenced by biological factors, and both are highly heritable. But there is a rather strong a priori argument against the likelihood of genetic influence on homosexuality. The argument is evolutionary: If homosexuality were heritable, one would expect it to be strongly selected against, and that

the responsible genes would long ago have vanished from the gene pool. This assumes that homosexuals reproduce less than heterosexuals, which indeed appears to be the case. Bell et al. (1981), for instance, found that white male homosexuals were much less likely to have been married than heterosexual males (26% versus 80%, respectively). Of those who married, homosexuals averaged about 0.9 children, compared to about 1.4 for heterosexuals. Thus, male homosexuals averaged about one-fifth the number of children through marriage compared to heterosexuals.

If this argument is correct, it supports the desirability of investigating environmental models of male homosexuality. The study reported below is a test of a promising environmental hypothesis. The hypothesis derives from experimental studies of animals, which are all environmental manipulations, and in this respect they are all potential vehicles.

Animal research regarding the etiology of homosexuality. Human subjects are the best research subjects for drawing inferences about human homosexuality. However, there are restrictions on research with humans which make research with nonhuman animals an important, complementary source of information. Most obviously, one cannot test a theory of the origins of homosexuality by inducing homosexuality in human subjects. One can, on the other hand, do this with animal subjects. Despite potential limits to generalizability, such research has proven influential in constructing hypotheses regarding the etiology of homosexuality.

One problem with interpreting studies using nonhumans relates to

usage of the term "homosexual." Beach (1977) has criticized the notion that male "homosexuality" in nonhuman animals is analogous to that in humans. Although he admits that homosexual behavior is fairly common among nonhumans, he notes correctly that such behavior alone does not justify the aforementioned definition of "homosexual." The definition requires a consistent preference for homosexual behavior. Furthermore, Beach argues that those rare naturally-occurring or experimentally-induced animals that show consistent homosexual behavior are not really homosexuals in the human sense. Rather, they have been feminized and are acting accordingly. This contrasts, according to Beach, with human homosexuality: "The stereotype cherished by an uninformed heterosexual public [is] . . . that homosexual men are expected to be effeminate. . . .The facts are quite the opposite. . . . "(ps 311-312) Evidently, Beach was unaware of the growing body of literature which supports the stereotypes (see above). Not only was Beach incorrect regarding the supposed lack of effeminacy in gay men, but the failure of his distinction supports the analogy between nonhuman and human homosexuality.

Though Beach's objection fails, one is not then entitled to use the term "homosexual" indiscriminately with nonhuman animals. Nor is one entitled to make easy generalizations from animal research to humans. The main point of animal research is to generate models of homosexuality and its etiology which can then be investigated in humans. In this respect, it does not matter whether or not we call the results of our experiments "homosexual" animals. In order to avoid unproductive controversy which such an appellation is liable to cause (see, for

example, Sigusch et al., 1982), I refer to relevant animals as "sexual inversions." Of course, the extent to which a given sexual inversion is similar to a human homosexual remains a highly pertinent question.

Ellis and Ames (1987) have summarized studies of experimentally-induced sexual inversions in non-human animals. They discuss five classes of relevant studies: (a) direct perinatal androgen manipulation, (b) pharmacologically blocking or augmenting of the perinatal effects of androgens, (c) induction of immunity responses to hormones involved in sexual differentiation, especially androgens, (d) sexual segregation during childhood, and (e) maternal and neonatal exposure to androgen-depressing emotional stress. Of these five, all except for (d) involve hormonal influence. Since I am mainly concerned with a model of male homosexuality which involves hormonal feminization, I do not discuss sexual segregation in the summary below. (Note: The most commonly used animals in the following studies are rodents, especially rats, although more complex animals such as rhesus monkeys have also been used. In the summary below it should be assumed that subjects are rats, unless otherwise specified.)

Direct perinatal androgen manipulation of males generally involves two distinct processes: castration soon after birth followed by artificial hormonal activation of puberty at an appropriate age. Male rats which have thus been inverted show little sexual interest in females and often display feminine mating postures in the presence of other males (Dorner & Hinz, 1968). Feminization following castration appears to occur only for those species whose neuro-organization is completed

postnatally. No such reaction occurs, for instance, in primates whose neuro-organization occurs entirely during gestation (Feder, 1984). This suggests that a neuroendocrinological model of human homosexuality should stress the effects (or non-effects) of prenatal hormones.

I have argued above that adrenogenital syndrome, a human female sexual inversion, is relevant to human male homosexuality. The animal inversion which most closely resembles adrenogenital syndrome is induced by injecting females with male-range doses of testosterone. Inverted female rodents show decreased displays of feminine receptive behavior (Ward & Renz, 1972), and often attempt to mount other females (Baum, 1976). In contrast to male inversions, castration has no effect on subsequent cross-gender behavior--testosterone appears to be the crucial variable. For reasons cited above, this suggests the possibility that male homosexuals may have been exposed to low levels of testosterone. It appears that female primates must be androgenized prenatally for significant inversions to occur (Phoenix, 1974), again implicating the prenatal period for human homosexuality.

Several drugs cause feminized inversions in male animals by interfering with the synthesis of androgens or blocking their effect in the brain. The most powerful drugs in this respect are the antiandrogens such as medroxyprogesterone (Depo-Provera) and flutamide. When these drugs are given to a pregnant mother while the fetuses are undergoing neuro-organization, male offspring are likely to be feminized in their sexual behavior (Neumann et al., 1970; Ward, 1972). The feminized behavior is identical to that described above for perinatal androgen

manipulation. However, there is an important difference between the two inversions which makes the drug-induced inversion especially interesting for human male homosexuality. In contrast to inversions caused by neonatal castration, rats feminized by prenatal exposure to antiandrogens have testosterone production within the normal male range (Hull et al., 1984, p. 1013). This fact supports the possibility that human male homosexuals could have feminized brains, despite the normal testosterone level typically found in gay males. Many other drugs may partially block masculinization of the brain during neuro-organization. Barbiturates, chlorimipramine, diethylstilbesterol (DES), pargyline, diazepam, marijuana, and reserpine all appear to have this effect.

Inversions have been caused in animals by inducing immune responses to sex hormones. For instance, an immunological response to testosterone was provoked in female rabbits who were later impregnated. Male offspring had feminized penises, although their testes were well-formed and functional. Unfortunately, sexual preference was not assessed in these rabbits (Bidlingmaier, Knorr, and Neumann, 1977). Kalcheim, Szechtman, and Koch (1981) induced an immune response to LHRH (lutenizing hormone releasing hormone) in male rats during the first 3 days after birth. On reaching sexual maturity these rats both mounted females and submitted to mounting by other males; i.e., they appeared to be analogous to human bisexuals.

The one remaining experimental manipulation for inducing inversions in nonhuman males is maternal stress during pregnancy (Ward, 1984). This is usually done by confining the mother to an intensely

lighted enclosure while simultaneously restraining her inside a narrow tube for a few hours per day. If this procedure is performed with a pregnant rat during its third trimester, male offspring exhibit varying degrees of inverted sexual orientation. Affected males submit to mounting by other males to an unusual degree, and often fail to mount receptive females, yet sex drive in these offspring is normal (Dahlof, Hard, & Larsson, 1977).

The physiological mechanism which appears to cause the stress-induced inversion is the elevation of stress hormones, such as cortisol, and of endogenous opiates, in the mother. Stress hormones are known to depress testosterone production in a variety of species (Bernstein, Gordon, & Rose, 1983). The mother's stress hormones cross the placenta and presumably affect the fetal production of testosterone. Ward has shown that injections of morphine to pregnant rats give rise to the same syndrome of atypical sexual behavior in male offspring as does prenatal stress. Furthermore, she demonstrated that if naltrexone, an opiate antagonist, is administered concurrent with prenatal stress, male offspring show a lesser degree of feminine sexual behavior than would otherwise be expected (Ward & Ward, 1985). Further support for the hypothesized mechanism is the observation that male fetuses whose mothers were stressed late in pregnancy have lower plasma testosterone levels than those whose mothers were not stressed (Ward, 1980). The maternal stress syndrome is associated with marked changes in the sexually dimorphic nucleus of the preoptic area within the preoptic hypothalamus (Anderson et al., 1986).

Though not an experimental manipulation, there remains one more relevant cause of sexual inversions in rats. Money and Ehrhardt (1972) describe work by L. G. Clemens, who found that the larger the number of brothers in a litter of rats, the more likely that sisters would exhibit masculine mounting behavior when primed with androgen in adulthood.

Implications of animal research for human homosexuality. To what extent do the paradigms outlined above recommend models for human male homosexuality? Alternatively, is it possible that processes analogous to those experimental manipulations help to cause human male homosexuality?

Although direct perinatal androgen manipulation successfully models some human sexual inversions (e.g., adrenogenital syndrome in females), this paradigm is not a plausible candidate for male homosexuality. It fails for obvious reasons: Male homosexuals have normal male gonads and normal male blood levels of testosterone. Direct androgen manipulation experiments support the possibility that the ineffective synthesis of testosterone, and subsequent incomplete masculinization of fetal brains is a final common pathway to male homosexuality. However, such experiments have nothing to say about why ineffective testosterone synthesis sometimes occurs.

In contrast, androgen-blocking drugs, immunological response to testosterone, and stress-induced suppression of testosterone remain possible etiological factors in human male homosexuality. All three hypotheses are similar in two respects: First, they all involve the induction of a substance in the mother which then passes through the

placenta--i.e., drugs, antibodies to testosterone, and stress hormones, respectively. Second, they emphasize feminization of the developing male fetus' brain due to inadequate testosterone synthesis or uptake (which is a consequence of the fetal reaction to the aforementioned substances). Because of these similarities, the hypotheses are not so much competitors as they are specific versions of a more general hypothesis.

Still, each of these hypotheses must be independently investigated in humans. Of the three, only the maternal stress hypothesis has so far been examined using human homosexuals as subjects.

Human evidence for the maternal stress hypothesis. Before examining human studies of the maternal stress hypothesis, let us briefly consider whether human physiology is of such a nature that one might reasonably expect the hypothesis to be correct for humans. In both monkeys and humans, stress appears to be associated with decreased testosterone production. However, in general, the adrenal response to stress in humans is much less dramatic than it is in animals (Sachar, 1980). Consequently, the stress hormones which suppress testosterone production are, on average, less concentrated during human stress reactions. One might then expect maternal stress to be less effective in inducing homosexuality in humans than in causing similar inversions in rat. On the other hand, there are marked individual differences among humans in hormonal responses to severe emotional stress (Mason, 1975). Perhaps some mothers secrete enough stress hormones so that their male offspring are particularly likely to become homosexuals. In this case, the maternal stress syndrome may be considered a case of the gen-

eral diathesis-stress model (Rosenthal, 1971), the diathesis being hormonal reactivity to stress, and the stress being random environmental events. Some implications of this model are considered in a later section. In summary, then, maternal stress-induced human homosexuality appears to be possible given our knowledge of human physiology, though one might expect the effect to be confined to certain unique individuals.

Three studies using human subjects provide some support for the maternal stress hypothesis. In the first, Dorner et al. (1980) surveyed a group of male homosexuals born in Germany between 1934 and 1953, and found that a disproportionately high number of them were born during and just after World War II. This would be expected from the maternal stress hypothesis, as these years in Germany must have been a time of great stress. Both the level of constant stress and the number of traumatic events which caused episodes of acute stress must have been markedly increased.

In a second study Dorner, Schenk, Schmiedel, and Ahrens (1983) attempted to assess the incidence of stressful events in the prenatal life of bisexual and homosexual men compared to heterosexuals. Homosexual and bisexual subjects more frequently reported that their mothers were exposed to stressful situations while pregnant with the subjects. Furthermore, the situations described by the bisexuals and homosexuals tend to be more severely stressful than those described by heterosexuals (e.g., father died during pregnancy versus father at war during pregnancy): For homosexuals, bisexuals, and heterosexuals, the reported incidences of moderate prenatal stress are 33%, 25%, and 6%,

respectively. The same figures for severe stress are 35%, 15%, and 0%, respectively.

Although these results are consistent with the maternal stress hypothesis, methodological problems of the study hinder its unqualified acceptance as support. One problem of the study is that the men, rather than their mothers, provided information about prenatal events. Though subjects were asked to consult their parents, it is unclear how extensively they did so. If some subjects failed to consult their parents, then their reports are based on their memories of events which their parents remembered and chose to tell them about. Experimental error is likely to be compounded for these subjects.

One might argue that the additional experimental error should work against confirmation of the experimental hypothesis, making the obtained results even more impressive. This would be the case if the error were assumed to be unsystematic. There is reason to believe, however, that it may not be. Homosexuals and bisexuals may feel a need to attribute causes for their sexual orientations that heterosexuals do not feel. They may be more prone to remember distinctive events which conceivably contributed to their difference. If so, differences in incidence of stressful events may reflect differences in memory rather than actual differences. Similarly, parents who are aware of their son's homosexuality may feel the same kind of pressure, and give the same biased data. That such retrospective bias occurs is illustrated by one study in which mothers of children with Down's syndrome recalled having experienced elevated stress during their pregnancies (Drillen &

Wilkinson, 1964). Of course, this is an extreme example--the birth of children with Down's syndrome is generally considered a tragedy--but it exemplifies a need to control for such bias.

Most recently, Ellis et al. (1988) tested the maternal stress hypothesis by contacting mothers directly to ask about their pregnancies with homosexual and heterosexual subjects. When the authors compared the stress reported by mothers of 39 male homosexuals to that experienced by mothers of 68 male heterosexuals, a marginally significant difference ($p < .05$, one-tailed test) in favor of mothers of homosexuals was found for the second trimester of pregnancy.

The study of Dorner et al. (1983), though it yields striking results consistent with the maternal stress hypothesis, fails to be conclusive for methodological reasons. In order to be conclusive, a similar study should at the very least include direct assessments of parents' (especially mothers') memories of stressful events, and try to control for the possibility of biased memories. Ellis et al. (1988) have gone this extra mile, methodologically, but their relatively small sample size precluded results which are statistically convincing. The study reported below is largely an attempt to provide these methodological improvements.

Maternal stress and the familiality of homosexuality. The probability that a hypothesis is veridical increases as the hypothesis survives various scientific challenges, such as direct experimental tests. A more subtle way in which this probability may increase is through the ability of the hypothesis to account for phenomena which it was not originally created to explain, and which are not easily

explainable without it. One phenomenon which might be somewhat more comprehensible in view of the maternal stress hypothesis is the familiality of homosexuality.

Research has generally shown a marked increase in the incidence of homosexuality in male siblings of male homosexuals (Bell, Weinberg, & Hammersmith, 1981; Pillard, Poumadere, & Carretta, 1982). The most thorough study to date (Pillard & Weinrich, 1986) yielded the following figures: About 20% of male siblings of homosexuals were homosexual or bisexual, compared to about 4% of the male siblings of heterosexuals.

What is the nature of this familiality? In other areas of behavior such as intelligence, personality, and psychopathology, familiality appears to be due mainly to heredity (Plomin & Daniels, 1987). I have discussed the difficulty in assuming that genetic variation underlies sexual orientation.

How might the maternal stress hypothesis explain the familiality of homosexuality? If some mothers are particularly vulnerable to stress and thus tend to secrete large amounts of stress hormones, their male offspring will have an increased probability of homosexuality. These mothers will be relatively likely to have more than one homosexual son. This model of homosexuality is the diathesis-stress model mentioned above. Unfortunately, the maternal stress hypothesis does not entirely circumvent the evolutionary issue raised above. One must still explain why some mothers are relatively vulnerable to stress. It would be surprising if such a vulnerability were not itself somewhat genetic, leaving us with the same paradox as before.

There is a strong prediction of the maternal stress hypothesis vis-a-vis familiality which appears to be confirmed in the limited available relevant data. The maternal stress hypothesis emphasizes the importance of the prenatal, intrauterine environment--the environment which the mother alone provides. Characteristics of the father are irrelevant. Thus, one would predict greater familiality on a homosexual's mother's side of the family than on his father's. Consistent with this expectation, Henry (1941) reported that 11 of 12 homosexual or bisexual aunts and uncles of homosexuals came from the maternal side. Similarly, Pillard and Weinrich (1986) found that both of the half siblings in their study who had Kinsey ratings above 0 were from the maternal side. Though these results are based on sparse data, they are quite intriguing. (It should be noted that these results are also consistent with a genetic hypothesis of X-linked recessiveness, assuming that none of the fathers of the probands were homosexual. In order to evaluate this possibility, it would be desirable to follow up sons of homosexuals.)

A related prediction is that among homosexuals, mothers' stress-proneness should be negatively correlated with stressful prenatal events. To see this, suppose that the induction of homosexuality requires that stress hormones reach a certain level, x, during a certain period of pregnancy. There are two factors which may increase the probability that this will occur: the mother's stress proneness (diathesis) and environmental stress (stress). The more the mother has of the former, the less the latter is needed, and vice versa. If we assume that familial homosexuality occurs when diathesis is strong, we can

make the following specific prediction: Mothers of multiple homosexual sons should have had fewer and less severe prenatal stresses than those with only one. Similarly, assume that we could identify a trait associated with the diathesis (e.g., emotionality). Such a trait should be especially elevated in those mothers who have given birth to more than one homosexual son. These hypotheses are tested in the study below.

Overview of the Study

Male and female homosexual and heterosexual subjects were recruited from the university population and from the population-at-large using homophile organizations. Kinsey behavior and fantasy ratings (described below), childhood gender conformity self-ratings, and ratings of the sexual orientation of siblings were obtained for each subject. In addition, the following were sent to the subjects' mothers: (1) a life events questionnaire, in order to assess the occurrence of stressful events in the year prior to and while the mothers were pregnant with the subjects; (2) a childhood personality rating scale with which to rate the subjects' childhood effeminacy; (3) a Hardiness questionnaire and an Emotionality scale (as measures of stress vulnerability) . The instruments described in (1) and (2) were completed by mothers, not only for subjects, but also for a heterosexual sibling, if one existed.

The following hypotheses were investigated:

1. Mothers of nonheterosexual (homosexual or bisexual) male subjects,

and mothers of male subjects who were effeminate during childhood (henceforth, effeminate males) should report, on average, more frequent and more severe stressful life events during the pregnancies of these subjects than should mothers of other subjects.

2. Mothers of nonheterosexual or effeminate males should report more frequent and stressful life events during the pregnancies of these subjects than during the pregnancies of heterosexual siblings.

3. Hypotheses 1 and 2 should not depend on parental awareness of offsprings' sexual orientation.

4. Stressful life events during mothers' pregnancies with male nonheterosexual or effeminate males should be elevated primarily during the second trimester of pregnancy.

5. Mothers of male nonheterosexual or effeminate males should have depressed hardiness scores relative to other mothers.

6. Male nonheterosexuals should report more nonheterosexual siblings than should other subjects. Within the nonheterosexual group, familiality should correlate positively with childhood effeminacy, Kinsey Fantasy scores, and mothers' Emotionality. Familiality should correlate negatively with mothers' Hardiness and with the amount of prenatal stress reported by mothers.

7. There should be higher rates of nonheterosexuality among maternal than among paternal uncles of male homosexuals and bisexuals.

Summary

Human male homosexuality is best considered a feminized deviation from typical masculine development. The cause is most likely biological; the best specific candidate is insufficient testosterone at the appropriate prenatal stage. One possible cause of low prenatal testosterone levels is maternal stress. The maternal stress hypothesis has received strong support from animal work, and has yielded promising results from human studies. We are now ready to delineate a study designed to test the maternal stress hypothesis, one which both corrects defects in designs of past studies and extends their scope.

Chapter 2

Method

Instruments

The following three questionnaires, which were answered only by subjects (and not by their mothers), may be found in Appendix 1.

Assessment of subjects' Kinsey ratings. Kinsey ratings for both fantasy and behavior were obtained for all subjects (Kinsey et al., 1948). For fantasy, a Kinsey "0" denotes a person whose sexual fantasies have always been of a heterosexual nature and never of a homosexual nature. A Kinsey "6" denotes a person whose fantasies have always been of a homosexual nature, and never of a heterosexual nature. The integers "1" through "5" signify gradations between these two extremes. The Kinsey scores for sexual behavior are precisely analogous to those for fantasy. The Kinsey Fantasy ratings served as the study's measure of sexual orientation.

Behavior Questionnaire. The Behavior Questionnaire assesses whether subjects' childhood behaviors were gender nonconforming. The five items on the questionnaire are adapted from Whitham (1977; Example: "Were you considered to be a sissy?"). The items were altered appropriately for female subjects. It was decided a priori to construct a scale adding all five items. Responses of "Don't know" are treated as intermediate to "Yes" and "No." Scale scores were obtained only for

subjects for whom all items were available. This scale is this study's measure of Self-Rated Childhood Gender Nonconformity (SCGN), or Self-Rated Childhood Effeminacy.

Relatives Questionnaire. The Relatives Questionnaire estimates the prevalence of homosexuality and bisexuality among subjects' brothers and uncles. Pillard and Weinrich (1986) report a close correspondence between homosexual subjects' knowledge of their sibings' orientations and the siblings' actual self-reported orientation. It seems likely that subjects' knowledge of uncles' orientation may be less accurate than their knowledge of siblings. In addition to uncles' orientations, subjects were asked how many uncles have never been married. This is probably more reliably assessed, and provides an independent, if far from perfect, indicator of homosexuality.

In addition to the prevalence of male homosexuality among relatives, the Relatives Questionnaire assesses the prevalence of female homosexuality and bisexuality among subjects' female relatives.

The following four questionnaires, which were answered by subjects' mothers, only (and not by subjects), may be found in Appendix 2. Appendix 2 also includes a copy of the letter sent to mothers.

Life Events Questionnaire. The Life Events Questionnaire yields the retrospective maternal reports of stress during their pregnancies used to test the maternal stress hypothesis directly. The Life Events Questionnaire assesses the occurrence of 28 potentially stressful life events as well as the degree to which each was stressful during the year before and the three trimesters of mothers' pregnancies with subjects.

Each mother was also asked to complete a questionnaire for both the child participating in the study and a heterosexual sibling of the subject, if available. The events were chosen from among those listed in the Psychiatric Epidemiological Research Interview (Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978). An event was chosen if (1) there was a reasonable chance that it might apply to women, and (2) there was a reasonable chance that it might be remembered for a long time. It was decided to make items fairly general (for example, "Marital problems" instead of "Marital infidelity") in order to achieve maximum coverage. Several events were added because they are especially likely to be true of pregnant women (e.g., "Unwanted pregnancy"). Two items about drug use were added in view of the aforementioned evidence that certain drugs may compromise testosterone synthesis. At the end of the questionnaire, space was provided for mothers to list pertinent events not explicitly listed in the questionnaire.

Each event was to be rated from 0 (zero) to 4, for each of four time periods: the year before pregnancy, the first trimester, the second trimester, and the third trimester of pregnancy. An additional cateogory was provided for instances in which mothers remembered that an event occurred during the pregnancy, but did not remember the specific trimester. An event was to be rated for any time period in which it caused stress, even if the actual event did not occur during the period. A "0" indicates that the event did not occur; a "1" indicates that the event occurred but caused no stress; a "2," "3," or "4" indicates that the event occurred and caused mild, moderate, or severe stress, respectively.

For each subject, and each sibling for whom mothers provided information, 18 stress composites were computed. The number of composites was determined by three different measures for each of six time periods. The three measures include the number of events rated as severely stressful ("4"), summed stress, and number of events rated greater than zero. The six time periods include the three trimesters of pregnancy. Since the timing of the hypothesized critical period of brain differentiation is inexactly specified, two additional time periods were investigated: the first and second trimesters combined, and the second and third trimesters combined. The final time period is throughout pregnancy. The latter includes those events which mothers could not place during a specific trimester.

The usefulness of life events questionnaires has been empirically supported (Thoits, 1983). Life events, as assessed by such questionnaires, are consistently associated with increased psychological disturbance--distress symptomatology, psychiatric hospitalization, and psychopathological behavior.

Personal Views Survey, and the EASI-Emotionality Scale. These scales serve as measures of maternal stress-proneness. (Actually, the Personal Views Survey is scored so that it is a measure of stress-resistance, the opposite of stress-proneness.) The Personal Views Survey is the short form of the composite measure of Hardiness employed by Kobasa and Puccetti (1983). The personality dimension, Hardiness, has been consistently found to decrease the effect of stressful life events in producing illness symptoms (Kobasa, 1979; Kobasa & Puccetti,

1983; Kobasa, Maddi, & Kahn, 1982). There are three related components to Hardiness: commitment, or the tendency to involve oneself in (rather than experiencing alienation from) whatever one is doing; control, or the tendency to feel and act as if one has an influence on the outcomes in one's life; and challenge, or the belief that change rather than stability is normal in life. The short Personal Views Survey has a reliability (Coefficient Alpha) of 0.86, and correlates 0.89 with the longer composite from which it is derived (Kobasa & Maddi, personal communication, November 1, 1982).

The Hardiness Scale has recently been the target of considerable criticism (e.g., Hull, Van Treuren, & Virnelli, 1987). Fortunately for the present study, the criticism concerns more the construct validity of Hardiness than its predictive validity vis-a-vis illness following stress. For instance, one criticism has been that Hardiness derives its predictive validity from being closely associated with neuroticism (Funk & Houston, 1987). Because of this concern, all subjects were given the Emotionality scale of the Adult EAS Temperament Survey (Buss & Plomin, 1984). This scale is essentially a neuroticism scale; its three subscales consist of Distress, Fear, and Anger.

It is important to note that the hypotheses regarding maternal stress-proneness concern mothers' stress-proneness during pregnancies with subjects; such pregnancies occurred some twenty years before the personality measures were administered. The five-year stability of the Hardiness scale is 0.61 (Kobasa & Maddi, personal communication, November 1, 1982).

Mother's assessment of subjects' gender conformity. Mothers were asked to rate subjects on eleven attributes of childhood personality. Of the eleven attributes, only four were of primary interest: masculinity, dominance, aggressiveness, and athleticism. These were embedded among the remaining seven traits in order to conceal the nature of the study, for practical and ethical reasons. In order to create scales of Maternally-Rated Childhood Gender Nonconformity, the eleven items were factor-analyzed separately for males and females. (Results are reported below.)

Subjects

Male and female homosexual and heterosexual subjects were recruited from several sources. Heterosexual subjects were enlisted from undergraduate psychology classes: an introductory class and a human sexuality class. Homosexual and bisexual subjects were also recruited from those classes; all students who indicated on a short questionnaire that they consider themselves homosexual or bisexual were contacted. Additionally, homosexual and bisexual subjects were obtained through campus Gay and Lesbian organizations at the University of Texas and several other universities. Homosexual and bisexual subjects were also enlisted through an advertisement in The Austin Chronicle, an alternative news magazine. In order to insure that subjects obtained this way would not differ excessively from those from the university, the ad specified that subjects must be under the age of

30 and have finished at least one year of college; however, one or the other requirement was waived several times.

Subjects were asked for permission to mail a questionnaire to their mothers. They were assured that their mothers would not be able to divine the exact nature of the study from the questionnaire; specifically, there was no hint in the questionnaire that the study has to do with sexual orientation. Subjects were encouraged to look at a sample questionnaire in order to reassure themselves of its innocuous appearance. Subjects could at any time refuse to continue participation.

Table 1 contains the frequency distribution of Kinsey fantasy ratings for subjects of each sex.

For reasons discussed below, subjects were divided a priori into four discrete groups: male nonheterosexuals (This group includes both homosexuals and bisexuals), male heterosexuals, female nonhetero-sexuals, and female heterosexuals. The criterion for inclusion among the nonheterosexual groups is a Kinsey fantasy score of 3 or greater. (A Kinsey score of 3 indicates a perfectly bisexual orientation: equal sexual interest in both sexes.)

The male nonheterosexual subsample consisted of 116 individuals with an average Kinsey fantasy score of 5.11 (SD=0.68). Eighty-three of the mothers of members of this subsample returned their questionnaires, a return rate of 83% of questionnaires mailed. (Of the male nonhetero-sexual group,10 subjects refused permission to send questionnaires to their mothers, 3 were adopted, and 3 mothers were deceased.) The female nonheterosexual subsample contained 25 females (mean Kinsey

Table 1

Frequency Distribution of Kinsey Fantasy Ratings, by Sex

Kinsey Rating	N (All Subjects) Males	Females	N (Mothers Responded) Males	Females
0	64	49	45	37
1	16	16	11	13
2	4	7	4	3
3	3	6	2	5
4	12	10	9	8
5	70	8	53	5
6	31	1	19	1
Totals	200	97	143	72

rating=4.16; SD=0.85). Nineteen of the mothers responded, a return rate of 83% of mailed questionnaires. (Two subjects refused permission to contact their mothers.) The male heterosexual subsample consisted of 84 subjects (mean Kinsey score=0.29; SD=0.55). 60 mothers returned questionnaires, a return rate of 71.4%. The female heterosexual subsample contained 72 subjects (mean Kinsey rating=0.42, SD=0.67). 53 mothers returned questionnaires, a return rate of 73.6%. The slightly higher return rate by the mothers of nonheterosexual subjects is probably due to an extra mailing to those mothers only.

Since the nonheterosexual subjects were selected from a less restricted population--in contrast to the heterosexual subjects, they did not come exclusively from psychology classes at the University of Texas--it is not surprising that they differ slightly from heterosexual subjects in their demographic backgrounds. Table 2 presents the available demographic data for the four groups. The nonheterosexual subjects are, on average, older than the heterosexual subjects and have less educated parents. When plausible reasons exist for believing that these differences may affect results, analyses will control statistically for these variables.

Hypotheses

The maternal stress hypothesis. The primary hypothesis of the study is that prenatal stress is a causal factor in human male homosexuality. Translated into the specifics of our investigation, we

Table 2

Means of Demographic Variables, by Sex and Orientation

Sex	Orientation	Mean Parental Education[a] $\bar{X}$	SD	Mother's Age $\bar{X}$	SD
Males					
	Heterosexuals (N=60)	4.96	1.29	47.1	6.6
	Nonheterosexuals (N=83)	4.52	1.52	51.6	8.1
Females					
	Heterosexuals (N=53)	4.92	1.21	46.8	7.3
	Nonheterosexuals (N=19)	4.68	1.08	48.7	6.9

[a]Parental education is the average of mother's and father's educational levels, which are computed as follows: 1=elementary or junior high school, 2=some high school, 3=graduated high school, 4=some college, 5=graduated college, 6=some graduate work, 7=graduate degree

expected that mothers of homosexuals would recall more stressful events during their pregnancies on the Life Events Questionnaire than would mothers of heterosexuals. Average reported stress should be particularly elevated during the second trimester of pregnancy. Average reported stress should be greater during mothers' pregnancies with homosexual subjects than during their pregnancies with heterosexual siblings of subjects.

Of significant concern in the present study is the fact that mothers were being asked to recall events which happened approximately 20 years ago. Uhlenhuth (1977) has shown that recall of even important events may be unreliable. To the extent that this is true, it should dilute any true differences between mothers of nonheterosexuals and those of heterosexuals, assuming that error of recall is random. If this is the case, the findings reported by Dorner et al. (1983) mentioned above are even more impressive.

However, the possibility that mothers of homosexuals are more likely to recall stressful events during pregnancy (whether or not the events actually occurred) complicates the interpretation of findings. It is possible that mothers who perceive their children as deviant may feel an unconscious need to attribute their child's behavior to negative events. The inclusion of lesbians as subjects in the present study was motivated by the need to control for this possibility. There is reason to suspect that prenatal stress is not a cause of female homosexuality. Hormonal theories of lesbianism posit an excess of prenatal testosterone (Ellis & Ames, 1987). Hence, lesbians comprise a group which may be

seen as deviant by their mothers, but have probably not experienced elevated levels of prenatal stress. Another safeguard against this possibility is to check whether mothers who know about their sons' homosexual orientations responded differently from mothers do not know. Unless the two groups are otherwise distinct, there should be no difference between them in the amount of prenatal stress reported by their mothers.

While one must not be unduly optimistic about the recall of events during pregnancy after 20 years, it seems likely that there are certain events (e.g., the death of a loved one), which are quite accurately remembered. The frequency of these events, at least, should differ between mothers of homosexals and those of heterosexuals. Because of this, we believe that tests of the maternal stress hypothesis will detect an effect, if it is there. As noted above, at the very least, the present investigation is methodologically superior to that of Dorner, who found the original effect for humans.

Familiality as an indicator of diathesis. Homosexuals are not randomly distributed across families -- apparently, some mothers are particularly likely to have homosexual sons. With respect to the aforementioned diathesis-stress model, such mothers may be thought of as high diathesis mothers. The higher the diathesis, the less stress is necessary to cause the condition of interest. The more a mother is prone to exaggerated hormonal reactions to stress, the less (objectively) stressful events need to be to raise cortisol levels sufficiently to impede testosterone production. Thus, it is expected that mothers of more than one homosexual should recall, on average, less stress during

their pregnancies than will mothers of only one homosexual. If the diathesis could be directly measured, mothers of more than one homosexual would be expected to have especially high scores. We attempt to investigate this possibility for two scales, Hardiness and Emotionality, which appear to be related to the hypothesized diathesis, stress-proneness. This aspect of our study is discussed more fully in the following section.

Another hypothesis regarding familiality is motivated by the consequence of the maternal stress hypothesis that the mother, and not the father, is important in the genesis of homosexuality. Assume that the diathesis, stress-proneness, is to some extent genetic. It follows that there will be an increase in homosexuality on a homosexual's mother's side, but not necessarily on his father's. Thus, we predicted that there would be a greater elevation of homosexuality among maternal than paternal uncles of homosexual subjects.

Hardiness. Presumably, hardy individuals are resistant to illness in the face of potentially stressful life events because they react less physiologically to these events than do less hardy individuals. (The intervening psychological mechanisms which make this possible are irrelevant to our purposes here.) According to the maternal stress hypothesis, one would expect mothers of homosexuals to be low in hardiness. Furthermore, mothers of more than one homosexual son should be especially low in hardiness. Of course, it is of concern that mothers' hardiness scores will be assessed approximately twenty years after the pregnancies of interest. This delay will certainly dilute any real effect,

although the hardiness composite has shown a respectable stability correlation of 0.61 over a 5-year period (Kobasa, 1982).

Gender conformity. The prenatal stress syndrome (Ward, 1972; 1984) is characterized by the feminization of behavior, including feminine sexual behavior, of male offspring who had been stressed prenatally. As noted above, prehomosexual male children tend to be effeminate. These two facts suggest the possibility that behavioral effeminacy, generally, is worth exploring as a dependent variable. That is, heterosexual subjects who were effeminate as children may have been so for reasons similar to most homosexual subjects. In this case, one would expect mothers of the former would also report elevated levels of prenatal stress. Thus, whenever possible, Self-Rated and Maternally-Rated Childhood Gender Nonconformity will be examined alongside sexual orientation.

A Note on Data Analysis and Power

For the results presented below, within-sex analyses treat sexual orientation as a continuous variable. For instance, within males, the correlation of Kinsey fantasy scores and mothers' stress ratings will comprise one test of the maternal stress hypothesis. For the sake of statistical power, it will be useful to combine female and male heterosexuals whenever theoretically appropriate. There is no reason to think, for instance, that female and male heterosexuals should differ appreciably in severity of prenatal stress. Thus, a more powerful test of

the prenatal stress hypothesis would compare the mean stress of male homosexuals with that of male and female heterosexuals combined.

Translating Dorner's results into the metric discussed above, where moderately stressful events are rated "3" and severely stressful events are rated "4," the point-biserial correlation between the dichotomous variable sexual orientation (bisexual or homosexual versus heterosexual) and stress is 0.54. This translates into an effect size of about 1.3. Using just the male subjects whose mothers responded, the present study has an 80% chance to detect an effect half that of Dorner's at the .01 significance level; it has greater than a 95% chance to detect such an effect at the .05 significance level.

The results of Ellis et al. translate into an effect size between 0.33 and 0.39. Taking the smaller of these estimates, the present study has about a 50% chance of detecting the effect at the .05 significance level using just the male subjects. Combining female and male heterosexuals, this probability rises to over 60%. Using both the larger N and the larger effect size, the probability is almost 80% of detecting an effect.

Chapter 3

RESULTS

Childhood Gender Nonconformity and Sexual Orientation: Males

Gender nonconformity scales. Two scales were constructed to measure childhood gender nonconformity (CGN). First, a scale of self-rated childhood gender nonconformity (SCGN) was formed by adding the five childhood items of the Behavior Questionnaire. Scale scores were obtained only for subjects who answered all five items. Coefficient alpha for the scale was 0.63.

A scale was constructed for the mothers' ratings of subjects' childhood personalities, via factor analysis, as follows: A scree plot of eigenvalues from a principal components analysis suggested that 2 factors could be extracted; 3 factors had eigenvalues greater than 1.0. Principal factor analyses with oblique rotation were performed, extracting from 2 to 3 factors. The 2 factor solution was deemed more interpretable, and its factor pattern matrix is reproduced in Table 3.

The first factor appears to contrast dominant, masculine behavior with passive, effeminate behavior. "Extraverted," "aggressive," "active," "dominant," and "athletic" had high loadings on the first factor, while the bipolar adjective, "masculine-feminine" had a slightly lower loading. The second factor appears to contrast conformity and good adjustment with nonconformity and poor adjustment. Items with high loadings on

Table 3

Factor Pattern Matrix for the 2-Factor Solution of Maternal Ratings of Childhood Gender Nonconformity for Male Subjects[ab]

Item Description	Factor Loadings Factor I	Factor II
Extraverted	0.67	0.04
Aggressive	0.67	-0.11
High Activity Level	0.62	0.12
Athletic	0.54	0.13
Dominant	0.65	-0.23
Emotional	0.29	-0.35
Conforming	-0.13	0.52
Rigid	0.05	-0.39
Feminine	-0.30	-0.38
Well-Adjusted	0.19	0.62
Polite	-0.01	0.57
Sickly	-0.05	-0.29

[a]The factor pattern was obtained after an oblique rotation (promax).

[b]The correlation between the two factors was -0.14.

the second factor included "nonconforming," "rude," and "poorly adjusted;" "emotional," "rigid," "feminine," and "sickly" have slightly lower loadings.

Although the item "feminine-masculine" loaded approximately equally on both factors, the first factor appeared to be more relevant to gender nonconformity. For instance, the item "athletic" also loaded highly on the first factor, and childhood athletic behavior has been found to discriminate between homosexual and heterosexual males (Grellert et al., 1982). All items with loadings greater than 0.30 on the first factor were included, and their raw responses added. Scale scores were obtained only for subjects for whom all items were available. The scale was constructed so that higher scores represent more passive and effeminate responses, and will henceforth be designated "maternally-rated childhood gender nonconformity," or MCGN. The internal consistency reliability (alpha) of this six-item scale was 0.74.

Self-rated and maternally-rated childhood gender nonconformity correlated 0.29 ($p < 0.0005$); corrected for attenuation, this correlation is 0.49. The correlation between self-rated CGN and the scale constructed using items from the second factor of the above factor-analytic solution for males was slightly lower, 0.22, thus providing some small support for the choice of the first factor. The correlation between self-rated gender nonconformity and the item, "masculine" was -0.28.

Relationship between gender-nonconformity and sexual orientation. Table 4 contains the correlations among sexual orientation (Kinsey Fantasy and Behavior) and childhood gender nonconformity (self-

Table 4

Correlations Among Measures of Childhood Gender Nonconformity and Sexual Orientation (Male Subjects)

	Kinsey Fantasy	Kinsey Behavior	SCGN
Kinsey Behavior	0.93		
Self-Rated Childhood Gender Nonconformity (SCGN)	0.53	0.52	
Maternally-Rated Childhood Gender Nonconformity (MCGN)	0.39	0.43	0.29

All correlations based on a minimum of 180 subjects, except for correlations involving MCGN, for which the minimum is 128 subjects.

All correlations are significant at the .001 level.

and maternally- rated) for males. Appendix 2 contains the correlations among self-rated and maternally-rated items and scales. The correlation between Kinsey fantasy ratings and self-rated CGN is 0.54; for maternally-rated CGN the correlation is 0.39. (All p's < 0.0001.) Adjusting for the unreliability of the gender nonconformity scales, these correlations rise to 0.68 for SCGN, and 0.50 for MCGN. Maternally-rated childhood effeminacy correlates slightly lower with its self-rated counterpart than with either Kinsey Fantasy or Kinsey Behavior. The individual items which best correlated with Kinsey fantasy were having been regarded as a sissy during childhood and childhood preference for female playmates (self-ratings), and maternal reports of childhood non-athleticism and effeminacy.

Table 5 contains the mean scores for CGN items and scales, separately for heterosexual and nonheterosexual males. (Appendix 3 contains the frequency distributions for self-rated gender nonconformity items, separately for heterosexual and nonheterosexual males.) Both self-rated and maternally-rated CGN average about one (nonheterosexual) standard deviation higher for the nonheterosexual male subjects. The nonheterosexual standard deviation for self-ratings was about twice the standard deviation for heterosexual subjects; the effect size is, thus, twice as large when computed using the heterosexual standard deviation. (The decision to use the heterosexual standard deviation rather than the pooled standard deviation, when the two standard deviations differ is after Glass et al. (1981). They argue persuasively that if two SDs differ substantially, an effect size computed using a pooled SD is meaningless.

Table 5

Mean Childhood Gender Nonconformity Items and Scales
For Male Heterosexuals and Non-heterosexuals

Item/Scale Description	Heterosexuals $\bar{X}$	SD	Non-heterosexuals $\bar{X}$	SD	ES[a]
Self Report Ratings[b]					
Individual Items					
1. Regarded as a sissy	1.19	0.54	2.02	0.93	1.54****
2. Loner	1.47	0.84	1.99	1.00	0.62****
3. Wished to be a girl	1.11	0.41	1.52	0.87	1.00****
4. Preferred associating with girls	1.13	0.37	2.03	0.98	2.43****
5. Dressed in female clothing	1.33	0.71	1.69	0.95	0.51**
Scales					
Sum of Items (SCGN)	6.22	1.42	9.26	2.85	2.14****
Maternal Ratings[c]					
Individual Items					
1. Introverted	2.31	1.12	2.38	1.07	0.07
2. Passive	2.61	1.03	2.86	1.16	0.24
3. Low Activity Level	1.82	0.96	2.15	1.23	0.34
4. Nonathletic	2.08	1.50	3.66	1.20	1.05****
5. Submissive	2.40	1.03	2.73	1.15	0.32
6. Calm	2.97	1.12	2.77	1.19	-0.18
7. Nonconforming	2.35	1.22	2.45	1.17	0.08
8. Adaptable	3.55	0.99	3.57	1.13	0.02
9. Masculine	4.25	0.75	3.46	0.93	-1.05****
10. Poorly adjusted	1.23	1.21	1.64	1.08	0.34*
11. Rude	1.05	1.03	1.22	0.96	0.17
12. Healthy	4.18	0.93	3.73	1.23	-0.48*
Scales					
Effeminacy[d]	6.78	4.28	10.42	4.26	0.85****

[a]Effect Size = $(X_1 - X_2)/s$, where X_1 is the mean of the nonheterosexual group, X_2 is the mean of the heterosexual group, and s is the standard deviation of the heterosexuals (Glass, 1977).
*P<.05; **P<.01; ***P<.001; ***P<.0001; probabilities refer to the differences between the means of heterosexuals and non-heterosexuals.
[b]Self-rated items and scales are based on a minimum of 85 heterosexual and 114 non-heterosexual subjects.
[c]Maternally-rated items and scales are based on a minimum of 61 heterosexual and 82 non-heterosexual subjects.
[d]This scale was formed by adding items 1, 2, 3, 4, and 5, and subtracting item 9.

In contrast, the effect size using either SD is meaningful.) There is no marked difference between the heterosexual and nonheterosexual groups in the size of the MCGN standard deviations.

Figure 1 represents the mean SCGN for males of each Kinsey fantasy score. Results represented in Figure 1 support the aforementioned division of male subjects into two groups. There was a sharp increase in mean gender nonconformity between Kinsey 2's and 3's; moreover, there was not much variation among the mean scores within either group. Figure 2 represents the distributions of SCGN for nonheterosexual and heterosexual males, separately. Nearly 50% of the nonheterosexuals obtained scores higher than the highest scoring heterosexual. The larger variance for nonheterosexuals is also apparent in the figure.

Table 6 contains the correlations among sexual orientation and childhood effeminacy scales for male heterosexuals and nonheterosexuals, separately. There is considerably less regularity in relationships among variables for both groups separately than for both groups combined. Curiously, it appears that there has been more attenuation for the nonheterosexual males (i.e., there are fewer significant correlations), though the variance of sexual orientation is somewhat greater for that group than for the heterosexual males (0.46 to 0.30, respectively). This suggests the possibility that some individuals with Kinsey fantasy scores of 1 or 2 may be more appropriately considered bisexual.

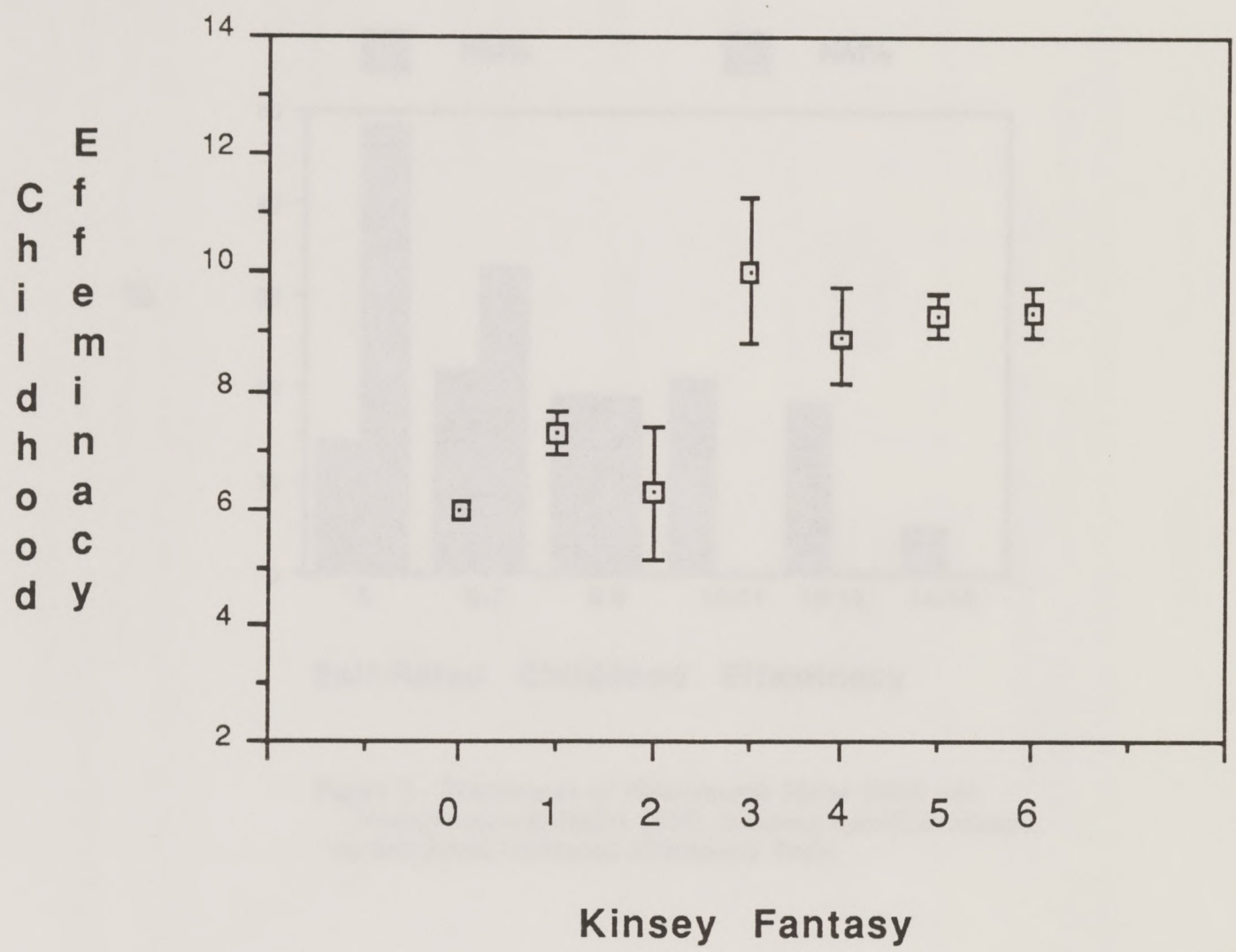


Figure 1. Mean self-rated childhood effeminacy (± 1 SE) for males of each Kinsey Fantasy Score.

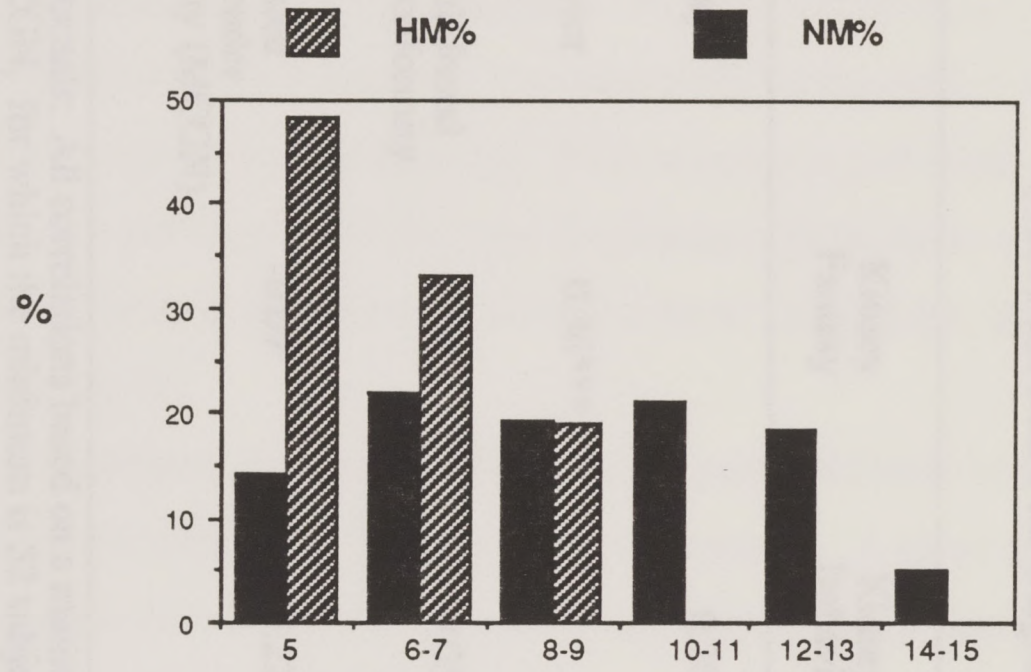


Figure 2. Percentages of Heterosexual Males (HM) and Nonheterosexual Males (NM) obtaining specified scores on Self-Rated Childhood Effeminacy Scale.

Table 6

Correlations Among Measures of Childhood Gender Nonconformity and Sexual Orientation: Heterosexual and Nonheterosexual Males (Above and Below the Diagonals, Respectively)

	Kinsey Fantasy	Kinsey Behavior	SCGN	MCGN
Kinsey Fantasy		0.20	0.25*	0.29*
Kinsey Behavior	0.36***		0.30**	0.12
Self-Rated Childhood Gender Nonconformity (SCGN)	0.00	0.05		0.13
Maternally-Rated Childhood Gender Nonconformity (MCGN)	-0.09	0.22	0.09	

Male Heterosexuals: All correlations based on a minimum of 74 subjects, except for correlations involving MCGN, for which the minimum is 52 subjects.
Male Nonheterosexuals: All correlations based on a minimum of 105 subjects, except for correlations involving MCGN, for which the minimum is 78.
*$\underline{P} < .05$; **$\underline{P} < .01$; ***$\underline{P} < .001$;

Childhood Gender Nonconformity and Sexual Orientation: Females

Gender nonconformity scales. As with the males, an attempt was made to construct two scales to measure childhood gender nonconformity (CGN) among the female subjects. First, a scale of self-rated childhood gender nonconformity (SCGN) was formed by adding the five female childhood items of the Behavior Questionnaire. Coefficient alpha for the scale was 0.72.

An attempt was made to construct a scale of maternally-rated childhood gender nonconformity via factor analysis. A scree plot of eigenvalues from a principal components analysis suggested that 2 factors could be extracted; 5 factors had eigenvalues greater than 1.0. Principal factor analyses with oblique rotation were performed, extracting from 2 to 5 factors. The two-factor solution was deemed most interpretable, and its factor pattern matrix is reproduced in Table 7.

Although the two-factor solution was the best obtained for female subjects, it was markedly less straightforward to interpret than that for males. Therefore, the solutions were given to five independent raters, consisting of one psychology professor and four advanced graduate students, to see if a reasonable consensus could be met regarding the factors' meanings. All five raters mentioned "extraversion" regarding the first factor, two added "activity," and two added a more evaluative descriptor ("social competence" and "adjustment," respectively). The second factor generated less conformity across raters. Two mentioned "rebelliousness," three mentioned synonyms for neurotic characteristics

Table 7

Factor Pattern Matrix for the 2-Factor Solution of Maternal Ratings of Childhood Gender Nonconformity for Female Subjects[ab]

Item Description	Factor Loadings Factor I	Factor II
Extraverted	0.67	0.03
Aggressive	0.67	0.34
High Activity Level	0.70	0.07
Athletic	0.68	-0.11
Dominant	0.26	0.70
Emotional	0.01	0.63
Conforming	0.19	-0.57
Rigid	-0.01	0.48
Feminine	-0.01	-0.09
Well-Adjusted	0.65	-0.33
Polite	0.36	-0.37
Sickly	0.17	0.11

[a]The factor pattern was obtained after an oblique rotation (promax).

[b]The correlation between the two factors was -0.17

("neuroticism," "emotionality," and "rigidity"), and two mentioned "dominance" (or "assertiveness").

In contrast to the male factors, it was unclear whether either of the female factors should be closely related to gender nonconformity. Curiously, the item "masculine" versus "feminine" loaded weakly on both factors. Therefore, for the following analyses, just that item was used for a measure of childhood effeminacy, since neither factor seemed appropriate. Self-rated childhood gender nonconformity correlated 0.43 with the 1-item scale of maternally-rated CGN (somewhat higher than the respective correlation for males).

Relationship between gender-nonconformity and sexual orientation. Table 8 contains the correlations among sexual orientation (Kinsey Fantasy and Behavior) and childhood gender nonconformity (self- and maternally-rated) for females. Appendix 2 contains the correlations among self-rated and maternally-rated items and scales for females. The correlation between Kinsey fantasy ratings and self-rated CGN is 0.53 ($p < 0.0001$). For the mothers' rating, the correlation was 0.32. All the self-rated gender nonconformity items correlated moderately well with Kinsey fantasy, with the exception of childhood preference for male playmates, which showed a weaker relationship. Among maternally-rated items, the strongest associations with sexual orientation were found for childhood dominance, nonconformity, masculinity, and poor adjustment.

Table 9 contains the mean scores for CGN items and scales, separately for heterosexual and nonheterosexual females. (Appendix 3

Table 8

Correlations Among Measures of Childhood Gender Nonconformity and Sexual Orientation (Female Subjects)

	Kinsey Fantasy	Kinsey Behavior	SCGN
Kinsey Behavior	0.84		
Self-Rated Childhood Gender Nonconformity (SCGN)	0.53	0.53	
Maternally-Rated Childhood Gender Nonconformity (MCGN)	0.32	0.53	0.42

All correlations based on a minimum of 87 subjects, except for correlations involving MCGN, for which the minimum is 66 subjects.

All correlations are significant at the .001 level.

Table 9

Mean Childhood Gender Nonconformity Items and Scales
For Female Heterosexuals and Non-heterosexuals

	Heterosexuals		Non-heterosexuals		
Item/Scale Description	$\bar{X}$	SD	$\bar{X}$	SD	ES[a]
Self Report Ratings[b]					
Individual Items					
1. Regarded as a tomboy	1.82	0.95	2.44	0.87	0.65**
2. Loner	1.25	0.67	1.88	0.97	0.94**
3. Wished to be a boy	1.32	0.73	1.88	1.01	0.77**
4. Preferred associating with boys	1.44	0.80	1.80	1.00	0.45
5. Dressed in male clothing	1.33	0.71	2.24	0.97	1.28***
Scales					
Sum of Items (SCGN)	7.12	2.34	10.24	3.38	1.33***
Maternal Ratings[c]					
Individual Items					
1. Introverted	2.31	0.90	2.00	1.37	-0.34
2. Passive	2.75	0.93	2.16	1.38	-0.63
3. Low Activity Level	2.06	0.89	2.00	1.25	-0.07
4. Nonathletic	2.38	1.27	2.68	1.89	0.24
5. Submissive	2.55	0.80	1.79	1.13	-0.95**
6. Calm	3.08	0.98	2.74	1.19	-0.35
7. Nonconforming	2.47	0.85	3.16	1.07	0.81**
8. Adaptable	3.60	0.93	3.05	1.22	-0.59
9. Masculine	1.94	0.86	2.68	1.05	0.81**
10. Poorly adjusted	1.23	0.87	1.79	1.13	0.59
11. Rude	1.40	0.88	1.42	1.02	0.02
12. Healthy	4.38	0.77	3.89	1.24	-0.64

[a]Effect sizes are computed so that positive numbers indicate that non-heterosexuals are higher in the trait as identified in the table.
*P<.05; **P<.01; **P<.001; probabilities refer to the differences between the means of heterosexuals and non-heterosexuals.
[b]Self-rated items and scales are based on a minimum of 72 heterosexual and 25 non-heterosexual subjects.
[d]Maternally-rated items and scales are based on a minimum of 51 heterosexual and 19 non-heterosexual subjects.

contains the frequency distributions for self-rated gender nonconformity items, separately for heterosexual and nonheterosexual females.) Both overall self-rated and maternally-rated CGN average about one (heterosexual) standard deviation higher for the nonheterosexual female subjects. The nonheterosexual standard deviation for self- and maternal-ratings of CGN was about one and one half times the standard deviation for heterosexual subjects; the effect size is, thus, somewhat smaller when computed using the nonheterosexual standard deviation.

Figure 3 represents the mean self-rated gender nonconformity for females of each Kinsey fantasy score. As with male subjects, there appears to be a discontinuous distribution of gender nonconformity. However, in contrast to males (for whom the break was between Kinsey 2's and 3's), the break was between Kinsey 3's and 4's. Figure 4 represents the distributions of SCGN for nonheterosexual and heterosexual females, separately.

Table 10 contains the correlations among the gender nonconformity and sexual orientation scales, separately for female heterosexuals and nonheterosexuals. For both subgroups, Kinsey Fantasy was strongly correlated with Kinsey Behavior scores, perhaps suggesting more continuity of meaning across the entire scale of sexual orientaion for females than for males. In light of the strong correlation between Kinsey Fantasy and Kinsey Behavior scores, it is somewhat curious that among nonheterosexual females, Kinsey Behavior correlates 0.74 with Maternally-Rated Childhood Gender Nonconformity, while the analogous correlation for Kinsey Fantasy is -0.02. There is no such difference for

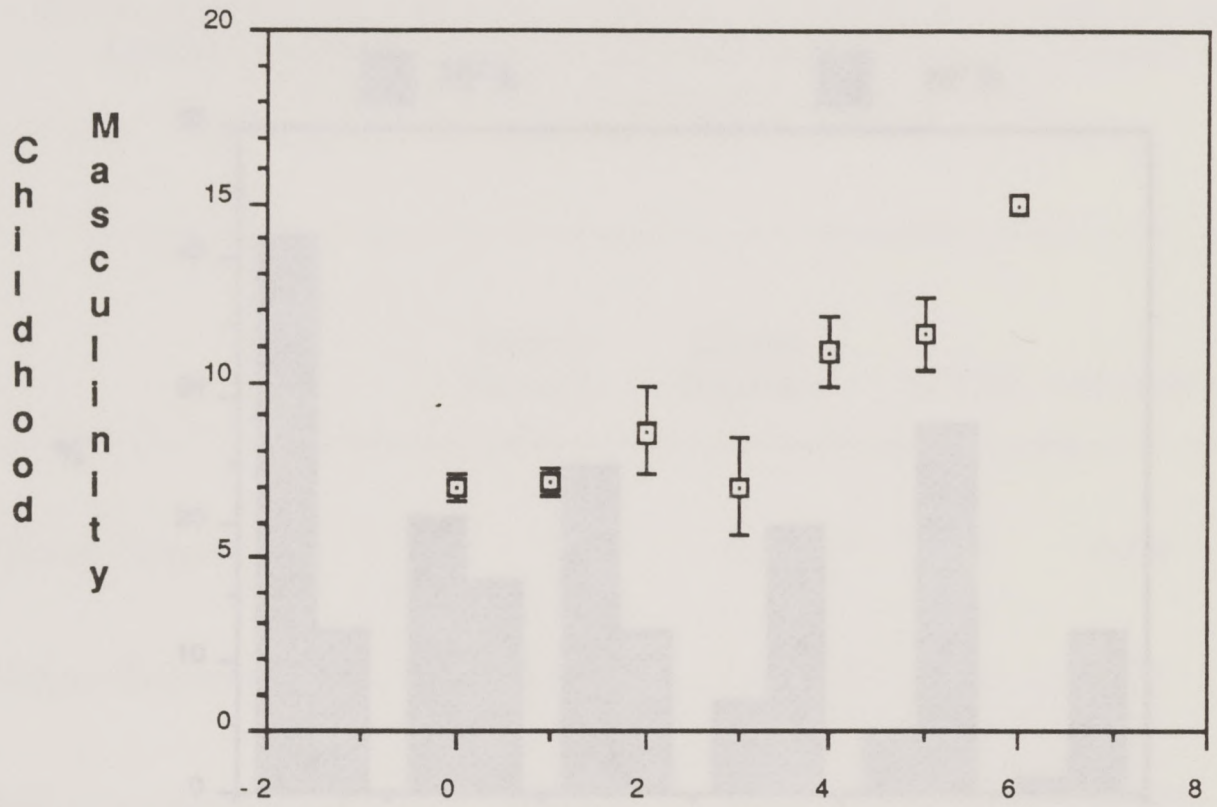


Fig. 3. Mean Self-Rated Childhood Masculinity (± 1 SE) for females of each Kinsey Fantasy Score.

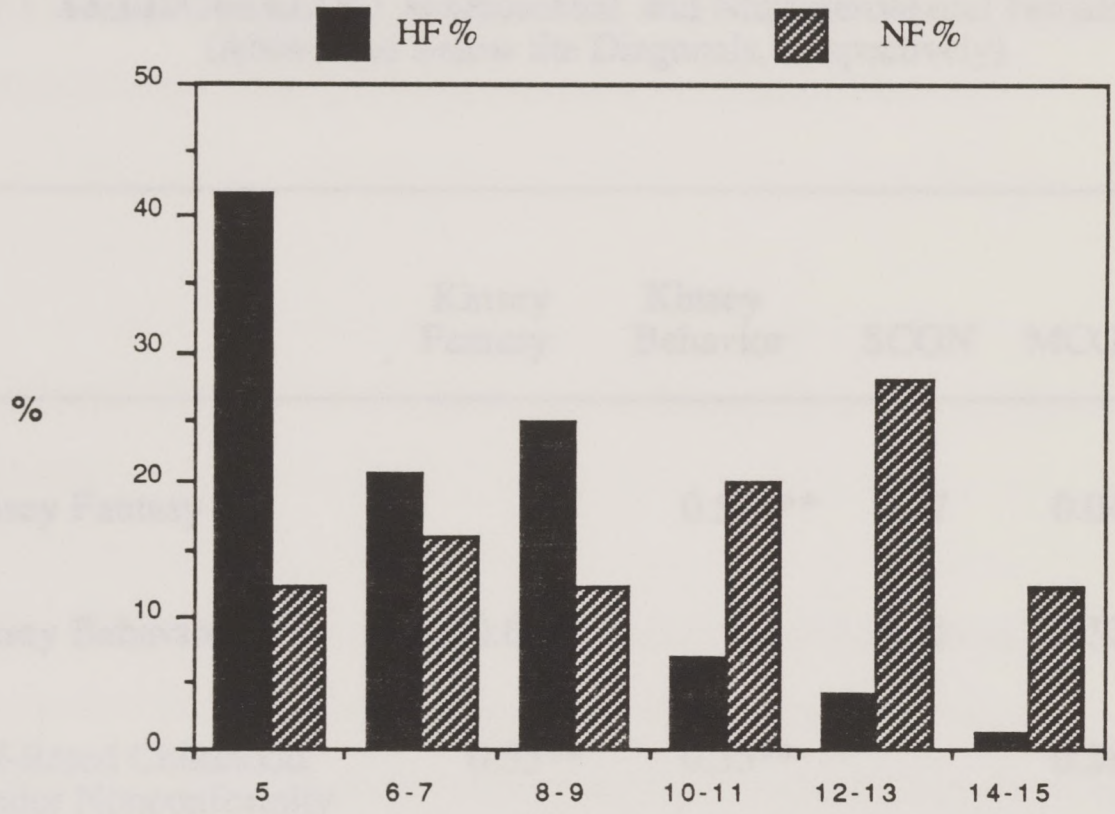


Figure 4. Percentages of heterosexual (HF%) and nonheterosexual females (NF%) obtaining specified scores on self-rated childhood masculinity scale.

Table 10

Correlations Among Measures of Childhood Gender Nonconformity and Sexual Orientation: Heterosexual and Nonheterosexual Females (Above and Below the Diagonals, Respectively)

	Kinsey Fantasy	Kinsey Behavior	SCGN	MCGN
Kinsey Fantasy		0.51***	0.17	0.04
Kinsey Behavior	0.61**		0.21	-0.32*
Self-Rated Childhood Gender Nonconformity (SCGN)	0.55**	0.55**		0.28*
Maternally-Rated Childhood Gender Nonconformity (MCGN)	-0.02	0.74	0.37	

Female Heterosexuals: All correlations based on a minimum of 64 subjects, except for correlations involving MCGN, for which the minimum is 48 subjects.

Female Nonheterosexuals: All correlations based on a minimum of 23 subjects, except for correlations involving MCGN, for which the minimum is 19 subjects.

*$\underline{P} < .05$; **$\underline{P} < .01$; ***$\underline{P} < .001$;

Self-Rated CGN; the respective correlations both equal 0.55. Given the small sample size for nonheterosexual females, and the fact that the different correlations are based on somewhat different samples, it is perhaps best not to speculate about such differences.

Childhood Gender Nonconformity and Sexual Orientation: Males vs. Females

For both males and females, self-rated childhood gender nonconformity was strongly predictive of adult sexual orientation. The primary difference between the results for males and females was in the structure of maternal ratings of childhood behavior. Factor analysis of ratings for males yielded two factors, the first reasonably interpretable as a gender conformity factor. A scale constructed from items loading on this factor correlated significantly with both self-rated childhood gender nonconformity and sexual orientation. Though two factors were also extracted from the data on females, neither was clearly identifiable with gender conformity -- primarily because the item "masculine" failed to show an appreciable loading on either. Thus, only the item "masculine" was used as a measure of maternally-rated childhood gender nonconformity for females.

Maternal Ratings of Prenatal Stress: Stress Measures

Table 11 contains the frequency distribution for the individual stress items. The entries represent the number of mothers who indicated that they had the specific experience during any of the specified periods.

Table 12 presents the means and standard deviations of the 18 stress composites examined in the following analyses.

Table 13 presents the correlations among three of the measures. Below the diagonal are the correlations across all subjects. The diagonal and above consist of correlations between subjects' scores and the scores of their siblings. Although both sets of correlations are uniformly quite high, all the correlations below the diagonal are higher than their respective correlations above it. Thus as should be expected, there is more continuity of stress within a pregnancy than between two pregnancies.

Regarding stress ratings of siblings, mothers of nonheterosexual females provided data for 74 siblings. The respective figures for nonheterosexual females, heterosexual males, and heterosexual females were 16, 53, and 45.

Maternal Ratings of Prenatal Stress and Orientation/Effeminacy: Males

Between-families. Table 14 contains the correlations over all males of the 18 stress composites with sexual orientation and

Table 11

Frequency Distribution and Mean Ratings of Stressors

Stressor	Frequency[a]	%	Mean Stress[b]	SD
Victim of a crime	7	3	2.71	1.25
Legal troubles	12	6	2.75	0.87
Change in pattern of schooling	30	14	1.93	1.14
Problems in school	10	5	2.80	1.23
Change in pattern of work or job	89	41	1.90	0.97
Problems in job	30	14	2.20	1.06
Moved residence	105	49	2.08	1.04
Pregnancy unplanned	90	42	2.27	1.23
Pregnancy unwanted	35	16	2.43	1.36
Pregnancy difficult	57	26	2.39	0.90
Marital problems	65	30	2.75	0.92
Problems with family members, friends	59	27	2.54	0.97
Away from spouse, extended time	52	24	2.52	1.04
Death of friend	6	3	2.83	1.17
Death of family member	27	13	2.63	1.28
Financial problems	87	40	2.22	0.98
Physical illness or injury	29	13	2.76	1.21
Illness or injury to family member	33	15	2.33	1.02
Mental illness	5	2	2.00	1.00
Mental illness to family member	10	5	2.50	1.18
Vaginal bleeding	31	14	2.19	1.19
Unmarried status	18	8	2.50	1.34
Automobile accident	20	9	2.05	0.94
Saw a counselor	7	3	2.00	1.00
Job loss to self or spouse	36	17	2.39	1.20

[a]Denotes the number of mothers who indicated that the event caused stress during one of the five time periods: Year before pregnancy, First trimester, Second trimester, Third trimester, and Sometime during pregnancy. Frequencies are computed for pregnancies with subjects, only (N=215). That is, mothers' ratings of pregancies with siblings are omitted.

[b]Mean stress was computed for the highest non-zero rated score for each stressor. (For example, if a mother rated a stressor as a "4" in one period and a "2" in another, "4" would be used in determining the mean.)

Table 12

Means and Standard Deviations of Composite Stress Ratings
Used as Dependent Variables
(N=215)

Time Period	Sum of Ratings		N Events Rated > 0		N Events Rated 4	
	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
First Trimester	5.91	8.33	2.60	3.23	0.44	1.37
Second Trimester	4.97	6.94	2.30	3.11	0.25	0.76
Third Trimester	5.36	7.03	2.40	3.06	0.33	0.80
First + Second Trimesters	10.88	14.74	4.90	6.18	0.70	1.90
Second + Third Trimesters	10.33	13.70	4.70	6.07	0.58	1.45
Throughout Pregnancy	18.31	22.95	8.19	9.67	1.17	2.66

Table 13

Correlations of Summed Stress for Three Time Periods
Within Subjects (N=215) and Between Subjects and Siblings (N=188)[a]

	First Trimester	Second Trimester	Third Trimester
First Trimester	0.61	0.58	0.52
Second Trimester	0.86	0.58	0.52
Third Trimester	0.83	0.92	0.49

[a]The diagonal and above consist of correlations between subjects and their siblings. Entries above the diagonal are computed as the average of relevant correlations.

Table 14

Correlations Between Stress and Orientation/Effeminacy: Males

Stress Composite	Orientation/Effeminacy: Kinsey Fantasy	SCGN[a]	MCGN
First Trimester			
Sum of Ratings	-0.06	-0.04	-0.00
N Events Rated > 0	-0.08	-0.08	0.03
N Events Rated 4	0.04	0.05	-0.00
Second Trimester			
Sum of Ratings	-0.02	-0.02	0.03
N Events Rated > 0	-0.02	-0.04	0.06
N Events Rated 4	0.11	0.07	-0.01
Third Trimester			
Sum of Ratings	-0.02	-0.06	0.03
N Events Rated > 0	-0.04	-0.04	0.08
N Events Rated 4	0.08	0.04	-0.03
First + Second Trimesters			
Sum of Ratings	-0.04	-0.03	0.01
N Events Rated > 0	-0.05	-0.06	0.04
N Events Rated 4	0.07	0.05	-0.00
Second + Third Trimesters			
Sum of Ratings	-0.02	-0.02	0.03
N Events Rated > 0	-0.03	-0.04	0.07
N Events Rated 4	0.10	0.04	-0.03
Throughout Pregnancy			
Sum of Ratings	-0.02	0.01	0.04
N Events Rated > 0	-0.03	-0.02	0.07
N Events Rated 4	0.09	0.07	0.00

[a]SCGN = self-rated childhood gender nonconformity; MCGN = maternally-rated childhood gender nonconformity.

All correlations are based on a minimum of 141 subjects.

effeminacy. Contrary to the maternal stress theory of male homosexuality, all the correlations between the stress composites and Kinsey fantasy and behavior scores, and childhood gender nonconformity are low and nonsignificant. The same pattern of results persists after equating statistically for mother's age, parents' education, and whether the mother spoke with the subject before filling out the questionnaire.

Table 15 presents the means and standard deviations for all stress composites separately for nonheterosexual males and all heterosexual subjects (males and females). Despite the additional power obtained by adding the females, there are still no significant results which would indicate that mothers of homosexual subjects experienced more prenatal stress than other mothers.

In order to see if a finer-grained analysis would yield any results consistent with the theory, correlations were computed between the individual stress items and orientation for male subjects. These results are presented in Appendix 4. There are 112 items, representing 28 stressors rated for four different time periods. Only three correlations are significant, and all are in the wrong direction. (The significant items are: problems in school, year before; used alcohol, year before; and used alcohol, first trimester.)

A final between-families analysis investigated whether the stress pattern across pregnancy differed between heterosexuals and nonheterosexual males. Recall that the hypothesized critical period is, roughly, the second trimester of pregnancy. One might, then, expect to see different patterns of stress for heterosexuals and nonheterosexual

Table 15

Mean Stress Composites by Orientation:
Heterosexuals (HS) Versus Nonheterosexual Males (NM)[a]

Stress Composite	Unadjusted Scores					Adjusted Scores				
	HS		NM			HS		NM		
	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]
First Trimester										
Sum of Ratings	6.0	8.3	5.5	8.7	-0.06	0.0	8.1	-0.3	8.0	-0.04
N Events Rated > 0	2.7	2.9	2.3	3.7	-0.11	0.0	2.9	-0.3	2.5	-0.16
N Events Rated 4	0.4	1.3	0.5	1.6	0.09	-0.1	1.2	0.1	1.6	0.13
Second Trimester										
Sum of Ratings	4.6	6.3	4.8	7.3	0.03	-0.4	6.2	-0.2	6.5	0.04
N Events Rated > 0	2.1	2.7	2.2	3.6	0.03	-0.1	2.6	-0.2	2.3	-0.02
N Events Rated 4	0.2	0.6	0.3	0.9	0.19	-0.1	0.6	0.1	0.9	0.21
Third Trimester										
Sum of Ratings	5.0	6.5	5.3	7.4	0.05	-0.4	6.3	-0.2	6.6	0.02
N Events Rated > 0	2.3	2.6	2.3	3.6	0.01	-0.1	2.5	-0.2	2.4	-0.06
N Events Rated 4	0.3	0.7	0.4	0.9	0.13	-0.1	0.7	0.1	0.8	0.14
First + Second Trimesters										
Sum of Ratings	10.6	14.2	10.3	15.5	-0.02	-0.4	13.8	-0.5	14.0	-0.01
N Events Rated > 0	4.8	5.4	4.5	7.2	-0.04	-0.0	5.3	-0.5	4.6	-0.09
N Events Rated 4	0.6	1.6	0.9	2.3	0.14	-0.2	1.6	0.2	2.3	0.18
Second + Third Trimesters										
Sum of Ratings	9.6	12.5	10.1	14.4	0.04	-0.8	12.2	-0.4	12.8	0.03
N Events Rated > 0	4.4	5.2	4.5	7.1	0.02	-0.2	5.1	-0.4	4.6	-0.04
N Events Rated 4	0.4	1.1	0.7	1.7	0.16	-0.1	1.1	0.1	1.7	0.19
Throughout Pregnancy										
Sum of Ratings	17.4	21.2	17.9	24.0	0.02	-0.9	20.8	-0.6	21.8	0.01
N Events Rated > 0	7.9	8.4	7.8	11.2	-0.01	-0.1	8.3	-0.8	7.7	-0.08
N Events Rated 4	1.0	2.2	1.4	3.2	0.16	-0.2	2.2	0.3	3.1	0.19

[a]All statistics are computed with the following minimum Ns: Heterosexuals, 113; Nonheterosexual Males, 80.

[b]Effect Sizes are computed so that positive values indicate that nonheterosexual orientation is associated positively with larger stress values for subjects.

Adjusted scores are residuals from the regression of stress on mean parental education, maternal age, and whether or not subjects were consulted before filling out the questionnaire.

males across trimesters. Testing this hypothesis was done via a repeated measures ANOVA, with stress as the dependent variable, trimesters as levels, heterosexuals versus nonheterosexual males as group, and stress during the year before pregnancy as a covariate. The specific hypothesis here would be the existence of a group x trimester interaction. Repeated Measures ANOVAs were performed for all three kinds of stress measures; none yielded either a significant interaction or a significant main effect for groups.

Within-families. Table 16 contains the correlations of sibling differences in the 18 stress composites with male subjects' orientation, effeminacy, and maladjustment. This analysis controls for mothers' tendencies to report similar levels of stress for siblings, suggested by the aforementioned substantial correlations between siblings' stress scores. For orientation and effeminacy, the pattern is the same: there are no significant correlations in the predicted direction; the lone significant correlation is opposite to that expected.

At first glance, there does appear to be one striking aspect to Table 16: The righthand column, correlations between stress and MCGN, contains only negative numbers. Multivariate analyses -- specifically, multiple regressions -- confirm that this consistency is due to the correlation among the different stress ratings; thus, their combined effect is nonsignificant.

Table 17, which contains the mean sibling differences for nonheterosexual males and for heterosexuals, gives essentially the same results--none of the mean differences is significant. Again, however,

Table 16

Correlations Between Sibling Differences in Stress and Orientation/Effeminacy: Males[a]

Stress Composite (Sibling Difference)	Orientation/Effeminacy		
	Kinsey Fantasy	SCGN[b]	MCGN
First Trimester			
Sum of Ratings	-0.03	-0.05	-0.15
N Events Rated > 0	-0.06	-0.05	-0.19*
N Events Rated 4	0.04	0.01	-0.01
Second Trimester			
Sum of Ratings	0.04	-0.03	-0.10
N Events Rated > 0	0.07	-0.00	-0.12
N Events Rated 4	0.05	-0.03	-0.00
Third Trimester			
Sum of Ratings	0.04	-0.04	-0.12
N Events Rated > 0	0.01	0.00	-0.15
N Events Rated 4	0.05	-0.06	-0.01
First + Second Trimesters			
Sum of Ratings	-0.00	-0.04	-0.13
N Events Rated > 0	0.01	-0.03	-0.17
N Events Rated 4	0.04	-0.01	-0.01
Second + Third Trimesters			
Sum of Ratings	0.04	-0.04	-0.11
N Events Rated > 0	0.04	-0.00	-0.14
N Events Rated 4	0.05	-0.04	-0.01
Throughout Pregnancy			
Sum of Ratings	0.01	-0.04	-0.13
N Events Rated > 0	0.04	-0.01	-0.16
N Events Rated 4	0.08	-0.00	-0.00

[a] All correlations are based on a minimum of 123 subjects.
[b]SCGN=self-rated childhood gender nonconformity; MCGN=maternally-rated childhood gender nonconformity.
*P < .05.

Table 17

Mean Sibling Differences in Stress Composites by Orientation: Heterosexuals (HS) Versus Nonheterosexual Males (NM)[a]

Stress Composite (Sibling Differences)	HS		NM		
	$\bar{X}$	SD	$\bar{X}$	SD	ES
First Trimester					
Sum of Ratings	0.4	7.9	0.9	5.8	0.07
N Events Rated > 0	0.2	2.6	0.3	2.1	0.02
N Events Rated 4	-0.0	1.6	0.2	1.4	0.15
Second Trimester					
Sum of Ratings	-0.6	6.1	0.8	5.5	0.23
N Events Rated > 0	-0.1	2.2	0.5	2.1	0.25
N Events Rated 4	-0.1	1.0	0.0	1.0	0.15
Third Trimester					
Sum of Ratings	0.0	6.8	1.4	6.0	0.21
N Events Rated > 0	0.2	2.4	0.5	2.3	0.15
N Events Rated 4	-0.1	1.1	0.1	0.9	0.17
First + Second Trimesters					
Sum of Ratings	-0.2	13.1	1.7	10.8	0.15
N Events Rated > 0	0.2	4.4	0.7	3.9	0.14
N Events Rated 4	-0.1	2.2	0.2	2.2	0.18
Second + Third Trimesters					
Sum of Ratings	-0.6	12.4	2.1	10.9	0.23
N Events Rated > 0	0.1	4.4	1.0	4.1	0.20
N Events Rated 4	-0.2	1.9	0.1	1.8	0.17
Throughout Pregnancy					
Sum of Ratings	-0.2	19.0	3.1	15.7	0.18
N Events Rated > 0	0.3	6.9	1. 6	6.3	0.18
N Events Rated 4	-0.3	3.5	0.4	3.1	0.20

[a]All statistics are computed with the following minimum Ns: Heterosexuals, 98; Nonheterosexual Males, 74.

[b]Effect Sizes are computed so that positive values indicate that nonheterosexual orientation is associated positively with larger sibling stress differences in favor of subjects.

there is a consistent pattern of low correlations -- this time positive (thus in the predicted direction). Again, multiple regressions confirm that this consistency is due to the correlations among the different stress ratings; thus, their combined effect is nonsignificant.

Maternal Ratings of Prenatal Stress and Orientation/Masculinity: Females

Between-families. Table 18 contains the correlations over all females between the 18 stress composites and sexual orientation, self-rated childhood gender nonconformity, and maternally-rated masculinity. In contrast to the results for males, there were several significant correlations between stress composites and sexual orientation, measured as Kinsey fantasy. All of the composites for which significant correlations were found implicate either trimester 2 or 3, though several include other trimesters as well. Essentially the same pattern of results obtained after equating statistically for mother's age and parents' education. It is notable that none of the stress composites correlated significantly with Kinsey behavior scores, though these scores have a correlation of 0.82 with Kinsey fantasy scores. The correlations were in the same direction, however, for Kinsey fantasy and behavior.

Table 19 presents the means and standard deviations for all stress composites separately for nonheterosexual females and all heterosexual subjects (males and females). There are significant differences between

Table 18

Correlations Between Stress and Orientation/Effeminacy: Females

Stress Composite	Orientation/Effeminacy: Kinsey Fantasy	SCGN[a]	MCGN[b]
First Trimester			
Sum of Ratings	0.05	-0.07	0.06
N Events Rated > 0	0.13	-0.11	0.03
N Events Rated 4	-0.09	-0.10	0.15
Second Trimester			
Sum of Ratings	0.25*	-0.07	-0.10
N Events Rated > 0	0.31**	-0.03	-0.05
N Events Rated 4	0.15	-0.13	-0.13
Third Trimester			
Sum of Ratings	0.22	-0.06	-0.08
N Events Rated > 0	0.31**	-0.02	-0.08
N Events Rated 4	-0.01	-0.12	-0.07
First + Second Trimesters			
Sum of Ratings	0.16	-0.07	-0.02
N Events Rated > 0	0.23*	-0.06	-0.01
N Events Rated 4	0.01	-0.11	0.04
Second + Third Trimesters			
Sum of Ratings	0.24*	-0.06	-0.10
N Events Rated > 0	0.31**	-0.03	-0.07
N Events Rated 4	0.07	-0.14	-0.11
Throughout Pregnancy			
Sum of Ratings	0.18	-0.03	-0.05
N Events Rated > 0	0.25*	-0.00	-0.04
N Events Rated 4	0.01	-0.11	-0.01

[a]SCGN = self-rated childhood gender nonconformity;
[b]MCGN = maternally-rated childhood gender nonconformity, which for females consists solely of the item "masculine-feminine."
All correlations are based on a minimum of 72 subjects.

Table 19

Mean Stress Composites by Orientation:
Heterosexuals (HS) Versus Nonheterosexual Females (NF)[a]

Stress Composite	Unadjusted Scores					Adjusted Scores				
	HS		NF			HS		NF		
	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]
First Trimester										
Sum of Ratings	6.0	8.3	6.9	7.2	0.12	0.0	8.1	1.2	7.6	0.16
N Events Rated > 0	2.7	2.9	3.3	2.6	0.22	0.0	2.9	0.8	2.7	0.24
N Events Rated 4	0.4	1.3	0.3	1.1	-0.12	-0.1	1.2	-0.2	1.2	-0.07
Second Trimester										
Sum of Ratings	4.6	6.3	8.0	8.6	0.42	-0.4	6.2	3.2	8.6	0.45
N Events Rated > 0	2.1	2.7	3.7	3.0	0.52*	-0.1	2.6	1.5	3.1	0.55*
N Events Rated 4	0.2	0.6	0.5	0.9	0.35	-0.1	0.6	0.2	0.9	0.37
Third Trimester										
Sum of Ratings	5.0	6.5	8.1	8.3	0.40	-0.4	6.3	2.9	8.4	0.40
N Events Rated > 0	2.3	2.6	3.7	3.0	0.49	-0.1	2.5	1.4	3.1	0.35
N Events Rated 4	0.3	0.7	0.4	1.0	0.09	-0.1	0.7	0.1	1.0	0.12
First + Second Trimesters										
Sum of Ratings	10.6	14.2	14.9	15.1	0.30	-0.4	13.8	4.4	15.5	0.32
N Events Rated > 0	4.8	5.4	6.9	5.4	0.40	-0.0	5.3	2.3	5.6	0.42
N Events Rated 4	0.6	1.6	0.7	1.7	0.09	-0.2	1.6	0.7	1.7	0.14
Second + Third Trimesters										
Sum of Ratings	9.6	12.5	16.1	16.8	0.40	-0.8	12.2	6.2	16.9	0.42
N Events Rated > 0	4.4	5.2	7.4	6.0	0.52*	-0.2	5.1	2.9	6.1	0.52*
N Events Rated 4	0.4	1.1	0.8	1.8	0.22	-0.1	1.1	0.3	1.7	0.27
Throughout Pregnancy										
Sum of Ratings	17.4	21.2	25.7	2.8	0.30	-0.9	20.8	7.9	28.3	0.32
N Events Rated > 0	7.9	8.4	11.8	9.7	0.42	-0.1	8.3	3.9	9.8	0.42
N Events Rated 4	1.0	2.2	1.2	2.9	0.08	-0.2	2.2	0.1	2.8	0.16

*P<.05.

[a]All statistics are computed with the following minimum Ns: Heterosexuals, 113; Nonheterosexual Females, 19.

[b]Effect Sizes are computed so that positive values indicate that nonheterosexual orientation is associated positively with larger stress values for subjects.

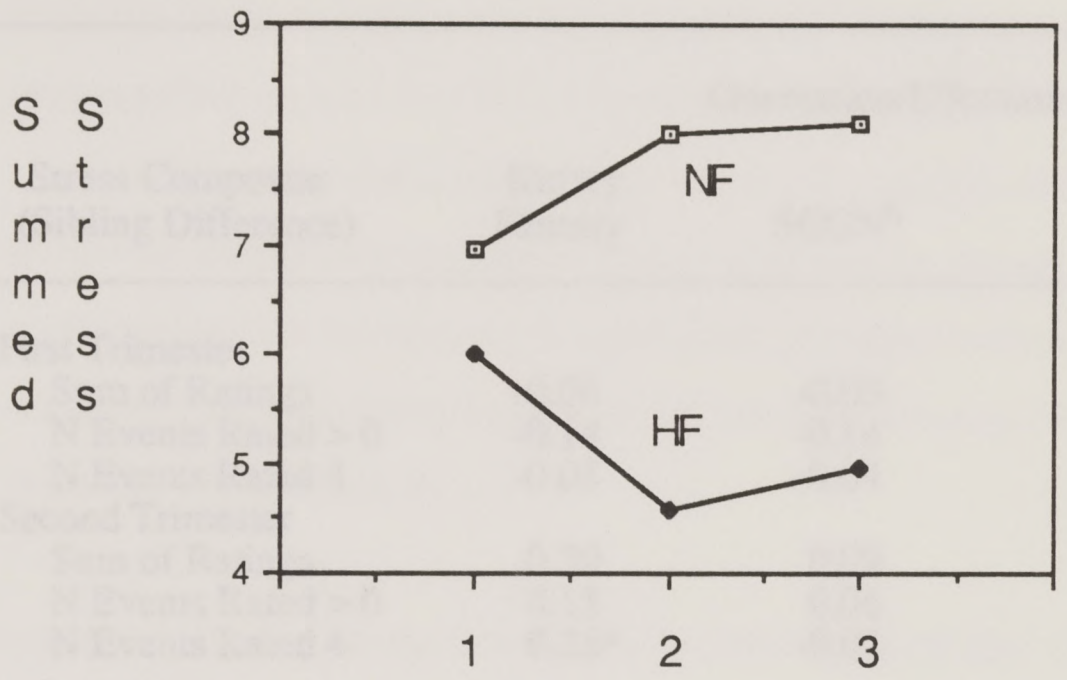
Adjusted scores are residuals from the regression of stress on mean parental education, maternal age, and whether or not subjects were consulted before filling out the questionnaire.

nonheterosexual females and heterosexuals for the number of events rated above 0 during the second trimester of pregnancy, and during the second and third trimesters of pregnancy combined.

Correlations between the individual stress items and orientation for female subjects are presented in Appendix 4. Several items are significant in the direction indicating that stress causes a nonheterosexual orientation: mental illness in a family member, first trimester; job loss to self or spouse, first, second, and third trimesters; and vaginal bleeding, sometime during pregnancy. Alcohol use, first, second, and third trimesters, also significantly predicted orientation, with mothers of nonheterosexual women using less alcohol.

As with the males, repeated measures ANOVAs were computed for female subjects in order to test for a trimester X group interaction. For females, this interaction was significant for both summed stress (F (1.4, 186) = 3.53, $p < 0.05$) and the total number of events rated (F (1.4, 188) = 4.63, $p < 0.05$). (Degrees of freedom are computed using the Greenhouse-Geisser correction.) The pattern of corrected means was the same for both variables; Figure 5 represents the means adjusted for the covariate, stress in the year before pregnancy, for summed stress.

Within-families. Table 20 contains the correlations of sibling differences in the 18 stress composites with female subjects' orientation, SCGN, and MCGN. There are two significant correlations between Kinsey fantasy scores and stress measures. Both significant correlations involve the number of events rated 4; this contrasts with the between-families analyses, in which the sum of ratings and events



NF = Nonheterosexual Females
HF = Heterosexuals

Figure 5. Interactions between group and trimester for summed stress. (Means adjusted for covariate, stress during year before pregnancy.

Table 20

Correlations Between Sibling Differences in Stress and Orientation/Effeminacy: Females[a]

Stress Composite (Sibling Difference)	Orientation/Effeminacy		
	Kinsey Fantasy	SCGN[b]	MCGN
First Trimester			
Sum of Ratings	-0.06	-0.03	0.19
N Events Rated > 0	-0.14	-0.14	0.17
N Events Rated 4	-0.05	-0.04	0.13
Second Trimester			
Sum of Ratings	0.20	0.09	0.17
N Events Rated > 0	0.15	0.06	0.20
N Events Rated 4	0.26*	-0.02	0.05
Third Trimester			
Sum of Ratings	0.14	0.11	0.11
N Events Rated > 0	0.07	0.03	0.11
N Events Rated 4	0.23	0.04	0.07
First + Second Trimesters			
Sum of Ratings	0.08	0.12	0.21
N Events Rated > 0	0.00	-0.04	0.21
N Events Rated 4	0.12	-0.04	0.12
Second + Third Trimesters			
Sum of Ratings	0.18	0.08	0.15
N Events Rated > 0	0.08	0.03	0.16
N Events Rated 4	0.26*	0.01	0.06
Throughout Pregnancy			
Sum of Ratings	0.11	0.05	0.19
N Events Rated > 0	0.05	0.05	0.19
N Events Rated 4	0.17	-0.01	0.10

[a] All correlations are based on a minimum of 61 subjects.
*P < .05.
[b] SCGN=self-rated childhood gender nonconformity;
MCGN=maternally-rated childhood gender nonconformity, which for females consists solely of the item, "masculine-feminine."

rated above zero yielded the significant correlations. Similar to the between-families results, the significant within-families results implicate the second and third trimesters of pregnancy.

When the additional male subjects were added, and mean within-families differences between all heterosexual males and nonheterosexual females were examined, two significant differences occurred, corresponding exactly to the two significant correlations just reported. (See Table 21.)

Maternal Stress Ratings and Subjects' Orientation/CGN: Males vs. Females

Contrary to the maternal stress hypothesis, higher maternal stress ratings did not predict a nonheterosexual orientation or childhood effeminacy among males. The hypothesis was confirmed neither between-families nor within-families.

In contrast, there were some results suggestive of a maternal stress effect for female nonheterosexuals: Some stress scores correlated positively and significantly with Kinsey Fantasy ratings. Some significant correlations occurred for both the between- and within-families analyses; those results implicate the second and third trimesters of pregnancy.

Table 21

Mean Sibling Differences in Stress Composites by Orientation: Heterosexuals (HS) Versus Nonheterosexual Females (NF)[a]

Stress Composite (Sibling Differences)	HS		NF		
	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]
First Trimester					
Sum of Ratings	0.4	7.9	-0.6	3.1	-0.24
N Events Rated > 0	0.2	2.6	-0.5	1.6	-0.40
N Events Rated 4	-0.0	1.6	0.0	0.0	0.02
Second Trimester					
Sum of Ratings	-0.6	6.1	1.9	7.4	0.35
N Events Rated > 0	-0.1	2.2	0.5	2.4	0.24
N Events Rated 4	-0.1	1.0	0.4	0.9	0.62*
Third Trimester					
Sum of Ratings	0.0	6.8	1.6	8.9	0.19
N Events Rated > 0	0.2	2.4	0.4	3.0	0.08
N Events Rated 4	-0.1	1.1	0.4	1.1	0.46
First + Second Trimesters					
Sum of Ratings	-0.2	13.1	1.3	10.0	0.14
N Events Rated > 0	0.2	4.4	0.0	3.8	-0.15
N Events Rated 4	-0.1	2.2	0.4	0.9	0.30
Second + Third Trimesters					
Sum of Ratings	-0.6	12.4	3.6	16.0	0.27
N Events Rated > 0	0.1	4.4	0.9	5.3	0.15
N Events Rated 4	-0.2	1.9	0.9	1.9	0.57*
Throughout Pregnancy					
Sum of Ratings	-0.2	19.0	2.9	18.4	0.17
N Events Rated > 0	0.3	6.9	0.6	7.5	0.04
N Events Rated 4	-0.3	3.5	1.0	2.2	0.38

[a]All statistics are computed with the following minimum Ns: Heterosexuals, 98; Nonheterosexual Females, 16.
[b]Effect Sizes are computed so that positive values indicate that nonheterosexual orientation is associated positively with larger stress values for subjects.
*$P < .05$

Maternal Stress-Proneness and Subjects' Orientation/CGN

Maternal Stress-Proneness Measures. Hardiness and EAS - Emotionality scales were formed by adding the appropriate items. Missing items were scored as intermediate responses; however, there was a limit of five missing items for Hardiness and 2 missing items for Emotionality. The internal consistency reliabilities of the hardiness composite and subscales were 0.79, 0.73, 0.66, and 0.88 for Challenge, Commitment, Control, and overall Hardiness, respectively. The reliability of the EAS Emotionality subscale was 0.83.

Table 22 contains the correlations among the personality scales used in the following analyses. The correlations are moderate to high, as would be expected if they share an underlying dimension of stress-proneness, or perhaps, neuroticism.

Relationship between hardiness and orientation/effeminacy: Males. A canonical correlational analysis was performed between full scale hardiness and EASI-Emotionality on one hand, and Kinsey Fantasy, self-, and maternally-rated childhood gender nonconformity on the other. The first canonical correlation was significant, and was primarily composed of both measures of stress-proneness and both measures of gender nonconformity.

Table 23 contains the univariate correlations between the measures of maternal stress-proneness and male subjects' sexual orientation and childhood gender nonconformity. There is a clear preponderance of low, significant correlations in the predicted direction, with low

Table 22

Correlations [a] Among Mothers' Personality Measures Related to Stress-Proneness

	Hardiness				EASI
	Challenge	Commitment	Control	Full Scale	Emotionality
Hardiness					
Challenge	(0.79)				
Commitment	0.58	(0.73)			
Control	0.49	0.64	(0.66)		
Full Scale	0.84	0.87	0.83	(0.88)	
EASI					
Emotionality	-0.36	-0.54	-0.28	-0.46	(0.83)

[a] Internal consistency reliabilities (alphas) in diagonals.
All correlations are based on a minimum of 214 subjects. All correlations are significant at the .001 level.

Table 23

Correlations Between Maternal Stress-Proneness and Orientation/Effeminacy: Males

	Orientation/Effeminacy		
	Kinsey Fantasy	SCGN[a]	MCGN
Hardiness			
Challenge	-0.08	-0.11	-0.21*
Commitment	-0.13	-0.19*	-0.29***
Control	-0.15	-0.16*	-0.22**
Full Scale	-0.14	-0.18*	-0.28***
EASI			
Emotionality	0.08	0.20*	0.17*

[a]SCGN = self-rated childhood gender nonconformity; MCGN = maternally-rated childhood gender nonconformity.
All correlations are based on a minimum of 139 subjects.
*P < .05; **P < .01; ***P < .001.

Challenge, Commitment, Control and Hardiness, and high Negative Emotionality predicting both homosexual orientation and childhood gender nonconformity. After adjusting for demographic variables, several of the significant correlations became nonsignificant. The correlations which remained significant were EAS Negative Emotionality with self- and maternally-rated childhood effeminacy (r's = 0.21, and 0.26, respectively; both p's less than 0.05); Challenge with maternally-rated childhood effeminacy ($r = -0.20$, $p < 0.05$); Commitment with maternally-rated childhood effeminacy ($r = -0.25$; $p < 0.01$); and Hardiness with maternally-rated childhood effeminacy ($r = -0.20$, $p < 0.05$). The consistency of these results is noteworthy, considering that the correlation between maternally- and self-rated effeminacy is less than 0.30.

Table 24 contains the means and standard deviations of the stress-proneness measures, separately for nonheterosexual males, and heterosexual subjects. There are only two significant differences: for challenge, and full scale hardiness. Both differences are in the predicted direction.

Relationship between hardiness and orientation/masculinity: Females. A canonical correlational analysis was performed between full scale hardiness and EASI-Emotionality on one hand, and Kinsey Fantasy, self-, and maternally-rated childhood gender nonconformity on the other. None of the correlations was significant.

Table 25 contains the univariate correlations between the measures of maternal stress-proneness and female subjects' sexual orientation and childhood gender nonconformity. None of the correlations

Table 24

Mean Maternal Stress-Proneness Measures by Orientation of Offspring: Heterosexuals (HS) Versus Nonheterosexual Males (NM)[a]

Stress Composite	Unadjusted Scores					Adjusted Scores				
	HS		NM			HS		NM		
	$\bar{X}$	SD	$\bar{X}$	SD	ES	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]
Hardiness										
Challenge	-8.7	7.0	-11.0	7.6	-0.31*	0.3	6.5	-0.8	7.0	-0.16
Commitment	1.6	4.8	0.7	4.9	-0.19	0.0	4.7	-0.3	4.6	-0.06
Control	-0.7	4.7	-2.0	5.3	-0.25	-0.1	4.7	-0.2	4.7	-0.03
Full Scale	-0.5	0.9	-0.7	1.0	-0.30*	0.0	0.9	-0.1	0.9	-0.10
EASI										
Emotionality	13.7	8.5	14.5	8.2	0.09	-0.1	8.6	0.6	8.0	0.09

*P < .05

[a] All statistics are based on a minimum of 79 mothers of nonheterosexuals and 113 mothers of heterosexuals. Adjusted scores are residuals from the regression of stress-proneness on mean parental education, maternal age, and whether or not subjects were consulted before filling out the questionnaire.

Table 25

Correlations Between Maternal Stress-Proneness and Orientation/Effeminacy: Females

	Orientation/Effeminacy		
	Kinsey Fantasy	SCGN[a]	MCGN
Hardiness			
Challenge	-0.03	-0.18	-0.09
Commitment	0.16	0.03	0.06
Control	0.11	0.15	0.13
Full Scale	0.09	-0.00	-0.02
EASI			
Emotionality	-0.08	-0.05	-0.06

[a]SCGN = self-rated childhood gender nonconformity; MCGN = maternally-rated childhood gender nonconformity--for females, this is the single item, "masculine."

All correlations are based on a minimum of 72 subjects.

approaches significance. Similarly, as Table 26 illustrates, there are no significant mean differences in maternal stress-proneness between heterosexuals and nonheterosexual females.

Maternal Stress-Proneness and Subjects' Orientation/CGN: Males vs. Females

In contrast to the analysis of maternal stress ratings which yielded significant results for females only, the analysis of maternal stress-proneness yielded significant results for males only. Specifically, both maternally-rated and self-rated childhood effeminacy were correlated with personality measures of maternal stress-proneness, suggesting that more stress-prone mothers tend to have more effeminate sons. The correlations were not significant for sexual orientation, though they were in the expected direction.

No significant correlations were obtained for females; no consistent pattern was observed.

The Effect of Maternal Knowledge on Responses

Subjects were asked to estimate their mothers' knowledge of the subjects' sexual orientation. Table 27 contains the frequency distribution of estimated maternal knowledge, for both males and females. A majority of the nonheterosexual subjects reported that their mothers were aware of their sexual orientation; this compares to

Table 26

Mean Maternal Stress-Proneness Measures by Orientation of Offspring: Heterosexuals (HS) Versus Nonheterosexual Females (NF)[a]

Stress Composite	Unadjusted Scores					Adjusted Scores				
	HS		NF			HS		NF		
	$\bar{X}$	SD	$\bar{X}$	SD	ES	$\bar{X}$	SD	$\bar{X}$	SD	ES[b]
Hardiness										
Challenge	-8.7	7.0	-8.4	4.8	0.05	0.3	6.5	1.2	5.3	0.16
Commitment	1.6	4.8	2.5	4.8	0.17	0.0	4.7	1.1	5.0	0.23
Control	-0.7	4.7	0.1	4.8	0.18	-0.1	4.7	1.1	5.0	0.25
Full Scale	-0.5	0.9	-0.3	0.8	0.16	0.0	0.9	0.2	0.9	0.25
EASI										
Emotionality	13.7	8.5	12.7	9.3	-0.12	-0.1	8.6	-1.27	9.0	-0.12

[a] All statistics are based on a minimum of 19 mothers of nonheterosexuals and 114 mothers of heterosexuals.

Table 27

Distribution of Mothers' Knowledge of Nonheterosexual's Sexual Orientation[1]

Level of Knowledge	Males N	Males %	Females N	Females %
Mother Does Not Suspect	5	(6)	4	(22)
Mother Might Suspect	23	(29)	4	(22)
Mother Definitely Suspects	2	(3)	1	(6)
Mother Definitely Knows	51	(63)	9	(50)

[1]Subjects' estimates of maternal knowledge; only nonheterosexual subjects whose mothers returned questionnaires are included here.

relatively few who asserted that their mothers had no suspicions.

Mothers were asked whether or not they had consulted with their children/subjects before filling out their questionnaire. Table 28 contains the correlations between maternal knowledge of subjects' orientation, whether or not mothers consulted subjects, and some variables of interest, for male nonheterosexuals. (The table is restricted to male nonheterosexuals as there are few female nonheterosexuals for whom complete data are available.)

Not surprisingly, maternal knowledge of subjects' orientation is correlated with the age of subjects. Maternal knowledge also correlates positively with Hardiness. If this represents bias, it is a bias which worked against the aforementioned results for males and stress-proneness, in which mothers of nonheterosexual males had lower Hardiness scores.

Mothers of nonheterosexual males who spoke with their sons before filling out their questionnaires reported higher summed stress over pregnancy than did such mothers who did not consult (r (73) = 0.25, p < .05). This alone would appear to indicate an effect of experimental demand. However, mothers of male heterosexuals showed an even stronger correlation (r (61) = 0.30 p < .05). Mothers of female heterosexuals showed a nonsignificant pattern in the same direction (r (53) = 0.11, p > .40). Thus, there appears to have been a general tendency for mothers who spoke to their participating children to report higher levels of stress.

Table 28

Correlations Among Nonheterosexual Males Between Mother's Knowledge of Subject's Sexual Orientation, Whether Mother Consulted Subject, and Other Variables

	Does Mother Know Subjects' Orientation?	Did Mother Consult Subject?
Subject's Age	0.40***	0.09
SCGN	0.14	0.13
Percentage of NH Brothers, Known or Suspected	0.02	-0.06
Sum of Ratings, Pregnancy	0.12	0.25*
Hardiness	0.29*	-0.10
EASI-Emotionality	-0.11	0.08

All correlations are based on a minimum of 73 subjects, except for the correlations concerning Hardiness, which are based on 62 subjects.

*$p < .05$; ***$p < .001$

Familiality of Nonheterosexuality: Male Nonheterosexuality

Siblings. Table 29 contains information provided by subjects about their siblings' sexual orientations. Male heterosexuals had 84 brothers, none of whom was either known or suspected to be nonheterosexual. Female heterosexuals had 60 brothers, 2 of whom are known to be nonheterosexual, and 1 of whom is suspected. Female nonheterosexuals have 30 brothers, 1 whom is known to be nonheterosexual, and 3 of whom are suspected. Male nonheterosexuals have 143 brothers, 15 of whom are known to be nonheterosexual, and an additional 15 of whom are suspected.

These rates are significantly heterogeneous for both known (Chi-square(3)=12.16, $p < .01$) and combined known and suspected (Chi-square(3)=25.8, $p < .001$) nonheterosexual brothers. The proportion of brothers known to be nonheterosexual was significantly greater for nonheterosexual males than that proportion for all other groups combined (Chi-square(1)=11.26; $p < .001$), as was the proportion rated either "known or suspected" (Chi-square(1)=21.89; $p < .001$).

In order to determine whether any subject characteristic predicts familial male nonheterosexuality, all nonheterosexual males with at least one brother were selected for analysis. For each subject, the percentage of nonheterosexual brothers was calculated as the number of brothers rated as nonheterosexual (known, suspected, or either, respectively) divided by the total number of brothers. (This controls for the fact that the probability of having a nonheterosexual brother is

Table 29

Familiality of Male Homosexuality and Bisexuality

Brothers	NM N	NM (%)	HM N	HM (%)	NF N	NF (%)	HF N	HF (%)
Total	143	(100)	84	(100)	30	(100)	60	(100)
Known Gay or Bisexual	15	(10)	0	(0)	1	(3)	2	(3)
Suspected Gay or Bisexual	15	(10)	0	(0)	3	(10)	1	(2)
Known or Suspected Gay or Bisexual	31	(21)	0	(0)	4	(13)	3	(5)

related to the total number of one's brothers.)

Table 30 contains the correlations between the percentage of nonheterosexual brothers with some variables of interest. Contrary to expectations, sexual orientation was not significantly related to familiality. There was one significant correlation involving childhood effeminacy: Maternally-rated effeminacy correlated -0.35 with the percentage of brothers suspected to be nonheterosexual. However, this variable correlated 0.14 with the percentage of brothers known to be nonheterosexual, and only -0.07 for the percentage of brothers either known or suspected to be nonheterosexual; thus, no consistent pattern emerged. The only other significant correlation was between the percentage of brothers suspected to be nonheterosexual with the summed stress score during pregnancy. This correlation, too, appears to be an isolated one. The correlation of summed stress with the percentage of brothers known to be nonheterosexual is -0.13; the correlation with percentage of brothers either known or suspected is only 0.05. Thus, there is no convincing pattern of correlations suggesting that any of the variables of interest predict familiality of male nonheterosexuality.

Given the fact that nonheterosexual males tend to be older than other subjects in the present sample, it is reassuring that there is only a small correlation between subject's age and familiality. This suggests that the age difference can account for very little of the substantial difference in rates of familiality between male nonheterosexuals and other groups. Still, given the relatively young age of many of the subjects, it is likely that some of their younger siblings are too young to

Table 30

Correlations Among Nonheterosexual Males Between Familiality[1] of Nonheterosexuality (NH) and Other Variables

Predictor	% Brothers Certain NH	% Brothers Possible NH	% Brothers Certain or Possible NH
		(N=71)	
Age	0.10	0.05	0.11
Kinsey Fantasy	-0.04	0.02	-0.02
SCGN	0.02	-0.03	-0.00
		(N=50)	
MCGN	0.14	-0.35*	-0.07
Hardiness	-0.16	0.05	-0.13
EASI: Emotionality	-0.10	0.05	-0.06
Summed Stress: Pregnancy	-0.13	0.31*	0.05

[1]Familiality is computed as the percentage of brothers in a family who fit the given criteria. Subjects without any brothers are excluded from the analyses.

*P < .05.

have manifested their sexual orientation in a detectable manner. Unfortunately, siblings' ages were not collected.

It is, however, possible to distinguish subjects who have only older brothers from those who have only younger brothers. Consider first subjects who have only older brothers. Of these subjects, nonheterosexual males had 66 brothers, 10 of whom were known to be nonheterosexual, and 5 of whom were so suspected. All other subjects with only older brothers had 67 brothers, 1 of whom was known to be nonheterosexual and 1 of whom was suspected. (For known nonheterosexual brothers, Chi-square(1) = 8.18, $p<.01$; for known and suspected nonheterosexual brothers combined, Chi-square(1) = 11.6, $p <$ .001.) Considering subjects with only younger brothers, nonheterosexual males had 48 brothers, 4 of whom were known to be nonheterosexual and 10 of whom were suspected to be. All other subjects with only younger brothers had 86 brothers, 2 of whom were known to be nonheterosexual, and 3 of whom were suspected to be. (For known nonheterosexual brothers, Chi-square(1) = 2.6, $p<.20$; for known and suspected nonheterosexual brothers combined, Chi-square(1) = 13.81, $p < .001$.) The clearest difference between subjects with only older brothers versus subjects with only younger brothers is that the former tend to have more confidence in their ratings of their brothers, being more likely to use the rating "known" nonheterosexual: Of the older brothers, 11 of 17 brothers with nonheterosexual ratings were "known" to be nonheterosexual; of the younger brothers, the respective figures were only 7 of 19 (Chi-square (1) =2.79, $p < .10$). The contrast between male nonheterosexual subjects

and all other subjects is also noteworthy. As brothers grow older, nonheterosexual males appear to become more certain of their nonheterosexual brothers' orientations. (4 of 14 nonheterosexual ratings of younger brothers were certain, compared to 10 of 15 ratings of older brothers; Chi-square(1)=4.21, $p < .05$) No such trend appears for other subjects. (3 of 5 nonheterosexual ratings of younger brothers were certain, compared to 1 of 2 ratings of older brothers; Chi-square (1) = 0.06, $p > .80$).

Uncles. Nonheterosexual males reported a total of 320 uncles; of these, 6 were known to be nonheterosexual and 26 were suspected. All other subjects reported a total of 496 uncles; of these, 7 were known to be nonheterosexual and 18 were suspected. The difference in proportions of known nonheterosexual uncles was nonsignificant (Chi-square (1) = 0.27); for known and suspected nonheterosexual uncles combined, the difference was significant (Chi-square(1)=7.36; $p < .01$).

Just taking uncles of nonheterosexual males, 166 were on the maternal side, versus 154 on the paternal side. Of the known nonheterosexual uncles, there were 3 on each side (Chi-square (1) = 0.01, N.S.). Of the suspected uncles, there were 13 on each side (Chi-square (1) = 0.04, N.S.). There were 22 unmarried uncles on the maternal side compared to 25 such uncles on the paternal side for nonheterosexuals (Chi-square (1)= 0.27, N.S.). Thus, contrary to expectations, there is no excess of nonheterosexual uncles on the maternal versus the paternal side for nonheterosexual subjects.

Familiality of Nonheterosexuality: Female Nonheterosexuality

Table 31 contains information provided by subjects about their siblings' sexual orientations. Male heterosexuals had 69 sisters, none of whom was either known or suspected to be nonheterosexual. Female heterosexuals had 59 sisters, 2 of whom are known to be nonhetero-sexual, and 4 of whom are suspected. Male nonheterosexuals have 132 sisters, 2 of whom are known to be nonheterosexual, and 11 of whom are suspected. Female nonheterosexuals have 24 sisters, 1 of whom is known to be nonheterosexual, and an additional 4 of whom are suspected.

These rates are not significantly heterogeneous for known nonheterosexual sisters (Chi-square(3)=2.99); they are significantly heterogeneous for both known and suspected nonheterosexual sisters, combined (Chi-square(3)=11.69, $p < .01$). Specifically, female nonheterosexuals have higher rates of nonheterosexual sisters, known or suspected, than the other groups combined (Chi-square(1)=5.2, $p < .05$).

Considering all subjects who have only older sisters, there were 103 such sisters, 5 of whom were known to be nonheterosexual, and 19 of whom were suspected to be nonheterosexual. For subjects with only younger sisters, the corresponding numbers are 100, 3, and 5, respectively. There is a clear tendency for older sisters to be suspected more frequently than younger sisters (Chi-square(1)=8.8, $p < .01$).

Table 31

Familiality of Female Homosexuality and Bisexuality

Sisters	NM N	NM (%)	HM N	HM (%)	NF N	NF (%)	HF N	HF (%)
Total	132	(100)	69	(100)	24	(100)	59	(100)
Known Lesbian or Bisexual	2	(2)	0	(0)	1	(4)	2	(3)
Suspected Lesbian or Bisexual	11	(8)	0	(0)	4	(17)	4	(7)
Known or Suspected Lesbian or Bisexual	13	(10)	0	(0)	5	(21)	6	(10)

Familiality of Nonheterosexuality: Male Versus Female Nonheterosexuality

Male nonheterosexuality provided a clearer pattern of familiality than did female nonheterosexuality. Both known and suspected nonheterosexual brothers were more common in the families of nonheterosexual male subjects than in families of other subjects. Nonheterosexual male subjects appeared to be less certain about nonheterosexuality in their younger brothers than in their older brothers; it may be that many of the brothers they suspected to be nonheterosexual will be confirmed as such when they become older.

Female nonheterosexuals reported an excess of sisters suspected--but not known--to be nonheterosexual. Older sisters are much more likely than younger sisters to be suspected of nonheterosexuality; this contrasts with male nonheterosexuality in which suspicion decreases (evidently turning into certainty) with brothers' age.

CHAPTER 4

Discussion

Childhood Gender Nonconformity

Males. Consistent with previous studies, a substantial number of homosexuals reported having been relatively gender conforming as children. For males, slightly less than half the nonheterosexual distribution failed to overlap the heterosexual distribution. This is similar to Bell et al.'s finding that over half of male homosexuals were typically "masculine" in development. For females, overlap was even greater.

Perhaps as striking as the mean differences between heterosexual and nonheterosexual subjects is the difference in their variances. The male nonheterosexuals had over four times the variance of male heterosexuals in total Self-rated Childhood Gender Nonconformity ($F(113, 84) = 4.03$; $p < .001$). This indicates substantial heterogeneity in the phenotypic development of nonheterosexual compared to heterosexual males. As noted earlier, this need not imply etiological heterogeneity. It is worth noting, however, that these results are consistent with both a disproportionate number of nonheterosexual males who fit the developmental stereotype, and a large number who do not. A successful etiological theory of male homosexuality must account for the phenotypic heterogeneity.

A unique feature of the present study is the collection and

subsequent analysis of mothers' retrospective ratings of subjects' childhood personality. The maternal data generally corroborate the view that heterosexuals and homosexuals differ in their childhood behaviors. For maternal ratings of males, the largest differences between these groups were on the items, "masculine" and "nonathletic" -- just where one would expect differences.

The difference between heterosexual males and nonheterosexual males is somewhat larger on the self-rated than on the maternally-rated scale, with approximate effect sizes of 2.0 and 1.0, respectively. This may be partly due to the inclusion in the latter of items which are not good measures of effeminacy (i.e., "introverted"). The decision to include these items was made on the basis of factor analysis. In hindsight, it might have been better to build a scale using an a priori notion of what should correlate with effeminacy.

The appreciable difference in effect sizes invites speculation as to whether mothers have been relatively unobservant or gay men may have overreported effeminacy. The question cannot be resolved with the present data; however one might suspect that gay men may be more accurate. In childhood, peers are the primary arbiters of who is gender nonconforming. Peers also mete out punishment for the nonconformity, and the punished child is surely more aware of this than his mother.

Taking just nonheterosexual males, childhood gender nonconformity did not correlate significantly with Kinsey Fantasy ratings. One interpretation of the failure to find such a correlation is that the latent trait of sexual orientation is scaled differently than the

Kinsey scale. The latter is not very informative about the former once scores exceed 2. For example, the true difference between a Kinsey 4 and a Kinsey 6 is much smaller than the true difference between a Kinsey 2 and a Kinsey 4.

Females compared to males. Results of this study are consistent with those of past studies with respect to the relationship between childhood gender nonconformity and adult sexual orientation. For both males and females, retrospective self-reports of childhood gender nonconformity -- i.e., effeminacy for males, masculinity for females -- are fairly predictive of an adult nonheterosexual orientation. It should be emphasized that the sampling scheme used in this study resulted in an overrepresentation of nonheterosexuals. This will tend to result in inflated correlations and effect sizes. On the other hand, for males, at least, high self-rated childhood gender nonconformity was exclusively associated with a nonheterosexual orientation. For both sexes, there was more predictability of orientation from high gender nonconformity scores than low scores.

The difference in the variances between heterosexuals and nonheterosexuals is about the same for females as for males; however, the ratio of variances is half that of males, owing to the larger values for the respective female groups. The variances of the two female groups are significantly different, (F (24, 71) = 2.09, $p < .05$), the nonheterosexual variance being larger.

Because of the small sample size of the nonheterosexual females, one must exercize caution in interpreting sex differences obtained in the

present study. Furthermore, since the items for female self-ratings were obtained by transforming the respective items for males (e.g., "tomboy" was substituted for "sissy"), the scales for the two sexes may not be directly comparable. Assuming for the moment that they are, the self-rated scale showed significantly more variance for heterosexual females than for heterosexual males ($F(71, 84) = 2.72$, $p < .001$). The effect size distinguishing female heterosexuals from female nonheterosexuals is about 60% of the corresponding effect size for males. These results conform with the notion that it is more acceptable for girls to show masculine behavior than for boys to be effeminate. Sissy is a pejorative term; tomboy is not. (Green, 1987).

Though the transformed self-rated items discriminate well between heterosexual and nonheterosexual females, the items showing the largest differences for males tend to show the smallest difference for females. The correlation between the effect sizes of the items for males and females was -0.90 ($p < .05$). This suggests that the relative importance of indicators of childhood gender nonconformity as predictors of nonheterosexuality may differ between males and females. Of course, this finding must be qualified by the small numbers of female nonheterosexuals and of self-rated items.

The factor solution of maternal ratings was somewhat different for females and males. This is broadly consistent with the results of Finn (under review), who factor analyzed items from several self-rated masculinity-femininity questionnaires. He found sizeable and stable differences between the sexes in the factor structure of masculinity-

femininity. However, because his study employed self ratings, and because he examined different item domains, his results are not directly comparable to those of the present study.

For maternal ratings of females, the largest differences were for "submissive," "masculine," and "nonconforming," with nonheterosexual females being less submissive, and more masculine and nonconforming. Sizeable, though nonsignificant effect sizes (nonsignificant due to the small N for nonheterosexual females) indicated that nonheterosexual daughters were perceived as having been less passive and adaptable, and more poorly adjusted, than were heterosexual females. This pattern is suggestive of tension between nonheterosexual daughters and their mothers. This is consistent with the findings of Bell et al. (1981), who examined the mother-daughter relationship from the daughter's point of view. These investigators found that homosexual females described their mothers in more negative terms than did heterosexual females.

Maternal Stress and Stress-Proneness

Males. Contrary to the primary hypothesis of this investigation, mothers of male nonheterosexuals reported no more stress, on average, than did mothers of heterosexuals. This negative finding contrasts sharply with the results of Dorner et al. (1983), whose appreciable effects for maternal stress have been noted earlier. It also contrasts -- though not so sharply -- with the smaller and marginally significant effect found by Ellis et al. (1988).

It is worth noting that although Ellis et al. purport to have found evidence for the maternal stress hypothesis, their results conflict more with those of Dorner et al. than with the negative results reported here. The effect size found by Ellis et al. -- somewhere between 0.33 and 0.39 -- is appreciably closer to the null hypothesis than it is to the effect size of approximately 1.30 found by Dorner et al. Furthermore, the effect sizes were computed with bisexuals included for Dorner et al.'s study but excluded for Ellis et al.'s study. If their inclusion or exclusion was made consistent across studies, the difference in effect sizes would increase, since both studies showed smaller effects for bisexuals.

There are at least three ways in which one might attempt to reconcile the present results with those of the earlier studies. One way is to take an essentially nihilistic position vis-a-vis this type of study: One can deny the possibility of obtaining valid results with a retrospective study. Mothers cannot be expected, one might argue, to be accurate in recalling events which occurred some twenty-odd years ago. Thus, why care that studies differ in their conclusions? None is trustworthy.

There are at least two responses which one can make to this argument: First, as noted earlier, the kinds of events "recalled" by Dorner's subjects -- e.g., being raped during pregnancy, being tortured by Nazis, and losing a husband -- are of such a severe nature that one can hardly fail to remember them. Thus, it is implausible that an effect of the size found by Dorner et al. (1983) would be obscured due to the admitted unreliability of retrospective reporting.

A second objection to the nihilistic position is that if

retrospective maternal reports of stressors during pregnancy were completely unreliable -- essentially random -- one would expect only random variation between studies. But the results of Dorner et al. contrast too sharply with those of the present study to be mere random variation. (The counterargument could be made that systematic errors in reporting may have led to significant, though invalid, differences.) Thus, an uncompromising nihilistic position is unconvincing.

One might attempt to reconcile the conflicting results of the three studies on the basis of their methodological differences. Methodologically, the study by Dorner et al. (1983) contrasts sharply with the study of Ellis et al. (1988) and the present study, which are quite similar. The former uses subjects' own reports, supposedly informed by mothers' knowledge. The latter studies use retrospective maternal reports. Primarily (though not exclusively) for this reason, the latter are superior methodologically. Other relative virtues of the latter include their attempt to hide the nature of the study from mothers and their inclusion of a list of events to cue mothers' recall. If one must choose between Dorner et al.'s study and the other two on methodological grounds, one must choose the latter.

If one makes this choice, one is arguably obliged to explain why Dorner et al.'s mothers might have systematically overreported stress. Constructing a plausible story is not difficult. However, due to the sparse nature of the authors' report, linking a plausible story with known facts is difficult. One does not know, for instance, what subjects or their mothers knew about the experimental hypothesis. It is possible

that they were responding to experimental demand; one cannot know from the relevant paper. It may be relevant that in East Germany (as in all countries in the Soviet bloc), where the subjects lived, homosexuality is seen as a character defect -- significantly more so than in the West. Thus, both subjects and their mothers may have felt conscious or unconscious pressure to "explain" subjects' sexual orientation.

It may not be necessary to choose between the present study and that of Ellis et al. I have already noted that the results of the two studies do not contrast sharply. Nevertheless, are there methodological grounds for choosing between them? Their respective methodologies are quite similar, up to the stress questionnaires constructed for mothers and the stress variables derived from the questionnaires.

Ellis et al. report results for three measures per period: (1) the number of stressful items endorsed, (2) mothers' general rating of stress during the period, and (3) the product of (1) and (2). At the very least, then, there were two nonsignificant results compared to one significant finding. Although Ellis et al. mention only predictions for the second trimester, Ellis and Ames had previously (1987) mentioned the first trimester as a possibility. Moreover, they tested for significance mean differences in 7 different time periods, of which at least three (the trimesters of pregnancy) could conceivably be important for sexual differentiation of the brain. Thus, the actual ratio of relevant, nonsignificant results to relevant significant results is probably appreciably greater than 2 to 1. This is important for hypothesis testing. The more relevant significance tests one performs (Here a "relevant significant

test" is one which one would be considered theoretically interesting if it yielded significant results.), the more tainted the uncorrected p values. Multiple significance tests are not a problem if, as in the present study, they are all negative. To the contrary, uniformly negative results despite a thorough battery of significance tests support the claim that no important effect is discernible. Multiple tests are more problematic if, as in the case of Ellis et al., only one is significant. One implication of this argument is that the difference between the results of the present study and those of Ellis et al. is even smaller than argued above.

Another difference between the two studies concerns the choice of subjects included for analysis. The marginally significant finding by Ellis et al. was obtained in the comparison of 68 male heterosexuals with 39 male homosexuals. Had 14 bisexual males and 134 female heterosexuals been added, the effect would have been slightly smaller, but it may have been significant due to the increased power. Thus, the difference probably cannot be attributed to choice of subjects; moreover, this difference fails to provide grounds for preferring one study to the other.

The inclusion of a within-family test in the present study presents both an additional hurdle and an additional opportunity for the maternal stress hypothesis. The fact that this test yielded no results consistent with the hypothesis is an additional hurdle unsuccessfully attempted; such a test was not performed by Ellis et al.

To conclude the methodological comparison of the three studies, the present study and that of Ellis et al. were both clearly methodologically superior to that of Dorner et al. Perhaps not

coincidentally, the former studies were also more like each other in their results, yielding effects not discernably different from zero (as in the present study), or only arguably so (as in Ellis et al.).

However, reconciling the conflicting results does not necessarily depend on savaging the methodology of Dorner et al. It is possible to believe the results of each study. Perhaps severe prenatal stress can cause male homosexuality. In times of severe stress -- such as World War II in Germany -- it becomes an increasingly common cause. But in contemporary America, severe stress is too rare to account for enough variance in sexual orientation to be detected reliably by a retrospective study. The implication that male homosexuals should be disproportionately conceived during times of great stress was supported, recall, by Dorner et al. (1980).

Besides accounting for the discrepant results across studies, this explanation may allow the resolution of conflicting results within the present study. Though this study yielded no significant differences between heterosexual and nonheterosexual males in the amount of prenatal stress experienced, there was an effect for maternal stress-proneness: Mothers of males who were effeminate as children were more stress-prone than mothers of gender-conforming males. This finding was predicted and is theoretically interesting -- but only if there is some reason to believe that maternal stress can cause effeminacy in male offspring.

Suppose, as suggested above, that prenatal stress can feminize brain development, but that sufficiently stressful events are normally

too rare to account for much variance in sexual orientation or childhood effeminacy. In this case, a more important determinant of the stress experienced by a mother might be her own stress-proneness. An analogous relationship may exist between intelligence and cultural deprivation. Extreme cultural deprivation can affect intelligence adversely; however, in contemporary America, such deprivation is so rare that the bulk of variance in intelligence is explained by genetic (and other, presently unspecified) differences (Clarke & Clarke, 1976).

There is reason to believe that in humans, individual differences in personality are important in determining physiological stress responses. In contrast to nonhuman animals, even severe stress does not guarantee a dramatic adrenal response in humans. Rats, for instance, exhibit marked corticosteroid responses under a variety of circumstances (Sachar, 1980). Near-maximal adrenal cortical activation can be produced in Rhesus monkeys through conditioned anxiety paradigms (Mason et al., 1957). While studies with humans have yielded the same general association between stressful situations and hormonal activation, human studies have shown considerable variability between subjects. It appears that humans exhibit varying degrees of psychological coping skills which can moderate the effects of "objectively" stressful events on hormonal secretion.

For instance, Wolff et al. (1964) studied thirty-one parents of children suffering from fatal illnesses. Throughout their extended crises, different parents showed characteristic levels of the hormone 17-OHCS, ranging from high to low excretion. The authors noted signifi-

cant and strong correlations between stress hormone secretion and "effectiveness of [psychological] defenses." The psychological characteristics were operationalized as follows: Low secretors --successful defenders -- were found (1) to demonstrate little or no overt distress, (2) to show little or no impairment of functioning in stressful situations, and (3) to demonstrate the ability "to mobilize further [their] defenses in superimposed acutely stressful experiences. . . ." (p. 581).

In a similar study Katz et al. (1970) studied 31 women awaiting biopsy of a breast tumor. They found a significant correlation between hydrocortisone production rates and "extent of defensive failing." Defensive failing was defined essentially as in Wolff et al. These two studies suggest that psychological stress-proneness may be more important than "objectively" stressful events in determining human stress hormone secretion.

But is the psychological stress-proneness found by those authors the same as the stress-proneness which yielded the significant correlation with childhood effeminacy in the present study? The former was framed in psychodynamic terms, while the latter was derived from trait-theory. Still, the behaviors listed above for "defensive failing" sound quite similar to items from a neuroticism subscale. For instance, unsuccessful defenders demonstrated much overt distress; this is similar to the EAS Emotionality item, "I frequently get distressed." Similarly the inability of the unsuccessful defender to hold up well under acutely stressful experiences is well-represented in the EAS item, "When I get scared, I panic." Thus, it seems reasonable to conclude that

the two different notions of stress-proneness are similar, and hence, that mothers scoring high on the stress-proneness subscales are more prone to excrete stress hormones than are low-scoring mothers.

Given the negative effect of some stress hormones on testosterone production, mothers high in neuroticism should be especially likely to have depressed testosterone production. Unfortunately for this hypothesis, Daitzman (1977) found a positive correlation between neuroticism and androgen levels in both human males and females. The correlation was significant, though the sample size for females was only 6.

Another problem with a hypothesis of stress-proneness causing childhood effeminacy or a homosexual orientation in males was noted above: Stress prone mothers presumably still require stressful events before they secrete stress hormones. However, maternal ratings of subjective stress failed to correlate with sexual orientation. Nevertheless, one could argue that the personality trait of stress-proneness is more stable than memories of subjective stress over a 25-year period. In any case, the finding of a relationship between effeminacy and mothers' stress-proneness needs to be replicated before explanations of it can be taken seriously.

Even accepting a positive correlation between maternal stress-proneness and childhood effeminacy, there are competing explanations. For instance, one possibility is that the correlation results from genetic transmission of neuroticism, which predisposes male offspring to be effeminate in childhood. In this case, the association between mother's neuroticism and offspring's effeminacy is not causal -- the more

important (and presumably larger) correlation is between the offspring's neuroticism and his own effeminacy. The problem with this interpretation is that neuroticism is barely familial (Carey & Rice, 1983). In order to yield the observed association between maternal neuroticism and childhood effeminacy, the correlation between offspring's neuroticism and effeminacy would need to be quite large. The available evidence -- personality data from male homosexuals -- have, in fact, shown small but significant differences between male homosexuals and heterosexuals (homosexuals > heterosexuals) in neuroticism (Manosevitz, 1971; Siegelman, 1972). This interpretation cannot be ruled out, though the lack of a strong theoretical rationale is surely not in its favor.

Another possibility which must be considered is that having an effeminate son makes a mother more neurotic. This cannot be ruled out in the present study. However, in principle, this possibility could be tested easily, if neuroticism scores were available from mothers, say, before subjects were born.

Fortuitously, there exist data which might be illuminating on some of these issues. The Texas Adoption Project (TAP; Loehlin, Willerman, & Horn, 1982) is an adoption study in which personality data were obtained from biological mothers of children adopted early in life, as well as from some adoptive parents. In particular, MMPI scores were obtained from 78 biological mothers and their adopted-away sons. Summing the Depression and Psychasthenia scales to form a neuroticism scale, and using the MF scale for effeminacy, the correlation between biological mothers' neuroticism and adopted sons' was 0.17 (J. C. Loehlin, personal

communication). Though not significant, this correlation is similar in magnitude to those obtained in the present study, and indicates that more neurotic biological mothers had more effeminate sons -- whom they have never met. The correlation between adoptive mothers' neuroticism and their biologically unrelated sons' MF scales is -0.05. Thus, it does not appear that the correlation between maternal neuroticism and subjects' childhood effeminacy can be explained as a reaction of mothers to their sons' personalities. So far, the results from the TAP are consistent with our preferred explanation.

Unfortunately, the correlation between adoptive mothers' neuroticism and the MF scales of their natural sons is -0.13, opposite what would be predicted by any of the aforementioned explanations. This suggests that the positive correlation may have been due to sampling error. On the other hand, the relevant N is relatively small -- only 46. Furthermore, there may be an additional reason for weighting the correlation between biological mothers and adopted-away offspring more heavily. The biological mothers have substantially higher mean Depression and Psychasthenia scores than those of the adoptive parents (Loehlin et al., 1982). Thus, there are more biological mothers obtaining scores in the upper range of neuroticism, which is where an effect on stress hormone secretion is most plausible. Regardless, the TAP data do not provide unambiguous support for a maternal stress-proneness effect. Moreover, the magnitude of the correlations obtained in both the present study and the TAP suggests that even if the effect is real, it is small.

The present study does not disprove the possibility that stress can

cause a nonheterosexual orientation in males, despite its uniformly negative results regarding this question. One cannot prove the null hypothesis. One can conclude with some confidence, however, that an effect the size of Dorner's is not general. If there is an effect of stress, it is a small one. This is consistent with the results of Ellis et al., who claim to have found a reliable effect; even if we accept their claim, the effect is small.

There are primarily two reasons why Dorner et al.'s 1983 study has been of interest vis-a-vis sexual orientation. First, because of the presumed mechanisms, it has been viewed as evidence for the neurohormonal theory of human male homosexuality. The failure of the present study to replicate Dorner et al. damages the neurohormonal theory only a little. The neurohormonal theory is arguably the only etiological theory of human male homosexuality currently viable. Furthermore, the maternal stress hypothesis is not straightforwardly derivable from the neurohormonal theory. It requires many ancillary hypotheses, e.g., that the human stress response is sufficiently intense to cause a decrease in testosterone production. There are undoubtedly more direct tests of the neurohormonal theory. The present study and that of Ellis et al. suggest that there are probably more promising tests, as well.

The second, and more important, reason why Dorner et al.'s study has been of interest to scientists studying sexual orientation was alluded to earlier: These results were of such a large magnitude that it appeared that a major cause of homosexuality -- a vehicle -- had been discovered. This was particularly important given the difficulty in

maintaining a genetic hypothesis for male homosexuality. The results of this study show that maternal stress is probably not such a vehicle.

Given that prenatal stress, if it causes male homosexuality, does not appear to be a common cause; and given that there are more direct tests of the neurohormonal theory of male homosexuality; this may be the rare instance in which we should conclude that additional research is not called for. Scarce resources would be better spent on the attempt to find "vehicles" for male homosexuality -- about which possibilities, more later.

Females. Surprisingly, and in contrast to males, there was a significant difference in reported maternal stress between nonheterosexual and heterosexual females. The effect appeared to involve primarily the second and third trimesters of pregnancy, and was found only for sexual orientation, not for gender nonconformity. Such an effect was found in both between- and within- families analyses.

The primary obstacle to taking the finding seriously is the lack of a theoretical explanation -- even post hoc -- of why it should have occurred. The maternal stress hypothesis seemed reasonable for human male homosexuality because stress hormones impede the production of hormones necessary for male sexual differentiation, and incomplete differentiation is the hypothesized cause of male homosexuality. No such scenario can be constructed presently for female homosexuality.

The second reason why the hypothesis seemed promising for human males is the aforementioned prenatal stress syndrome in rats (Ward, 1980), in which male offspring of females stressed during pregnancy

exhibit elevated rates of feminine sexual behavior. The effect of stress on female offspring has been studied by Herrenkohl and her colleagues. Prenatally stressed female rats are less fertile and fecund (Herrenkohl and Politch, 1978; Herrenkohl, 1979), and show disruptions of the estrous cycle and effects on sexual behavior (Herrenkohl and Politch, 1978; Herrenkohl and Scott, 1984; Politch and Herrenkohl, 1984). Prenatally stressed females showed increased sexual receptivity. There have been no reports of increased masculine sexual behavior in prenatally stressed females.

The finding is not altogether implausible. Knowledge about how stress affects the developing fetus is just beginning to accrue. Furthermore, the time periods implicated for females are precisely the time periods of interest. On the other hand, the fact that no association was found between maternal stress-proneness and female sexual orientation detracts from the finding's plausibility as an explanation of female nonheterosexuality. The lack of an explanation for the finding of increased prenatal stress among human female nonheterosexuals obliges one to replicate the finding before it is accepted as veridical.

Familiality

Male nonheterosexuality. Consistent with previous investigations (e.g., Pillard and Weinrich, 1986), the present study indicates that male homosexuality is familial. The proportion of brothers of male nonheterosexuals rated as either known or suspected nonheterosexuals,

21%, is strikingly similar to the figures obtained by Pillard and Weinrich, which ranged from 18% to 22%, depending on the analysis.

In contrast, only about 4% of brothers of other subjects were at least suspected to be nonheterosexual, a figure also quite similar to that obtained by Pillard and Weinrich. Two aspects of the figure for controls deserve mention. First, not one of the 84 brothers of male heterosexuals was rated as at least suspected nonheterosexual. Given a conservative rate for nonheterosexuality of 4% in the general population, 3 of the 84 brothers would be expected to be nonheterosexual. Given the fact that male heterosexuals also failed to suspect even one of their 69 sisters of nonheterosexuality, it is tempting to conclude that this group was rather uncurious about such matters. Second, the nonheterosexual females report a relatively high rate, 13%, of known or suspected nonheterosexuality in their brothers. This figure, however, is based on only 30 brothers, total. Furthermore, only 1 of the 4 brothers rated as nonheterosexual was known to be so; the others were suspected. Thus, it seems likely that the 0% and 13% rates for brothers of heterosexual males and nonheterosexual females, respectively, are anomolous; the former being an underestimate, the latter an overestimate.

One might question whether the familiality of nonheterosexuality has been demonstrated in the present study. Indeed, familiality was not assessed directly. One might argue that we have investigated "known familiality," and that this might be quite distinct from true familiality. For example, it is plausible that if one son has "come out," nonheterosexual brothers are encouraged to do the same. In this case,

the familiality found here might just represent the effects of a family atmosphere favorable to admitting a nonheterosexual orientation. However, there are at least two studies which cast doubt on this interpretation. As noted above, Pillard and Weinrich (1986) assessed familiality directly and found rates quite similar to those of this study. Secondly, Eckert et al. (1986) found two male pairs of monozygotic twins separated at birth in which at least one twin was homosexual. One of the pairs was clearly concordant; the other was neither clearly concordant nor discordant. Regarding studies of unseparated MZ twins reviewed by Eckert et al., over half the pairs in which at least one twin was homosexual were concordant for homosexuality. Thus, MZ twins have concordance rates higher than equivalent rates for siblings, and from the limited available data it does not appear that postnatal environment contributes much to their similarity. It does not seem unreasonable to proceed as if familiality of nonheterosexuality is an actual phenomenon.

Taking only the nonheterosexual male subjects, none of the variables examined convincingly predicted familiality -- i.e., which subjects would have a greater number of nonheterosexual brothers. This analysis was motivated by psychopathology research, in which claims are often made for "dosage effects." For instance, there appears to be a dosage effect for age of onset in schizophrenia -- schizophrenics who are younger at their first episodes tend to have more sick relatives than those who are older. The lack of a "dosage effect" for nonheterosexuality is consistent with the failure of sexual orientation to correlate highly with childhood effeminacy <u>within the nonheterosexual males</u>. Dosage

effects suggest multifactorial, polygenic causation. The absence of such effects leaves open the possibility of single major influences (e.g., major genes).

The "maternal effects" hypothesis specified that if mothers contribute to the nonheterosexuality of their sons (e.g., by being stress-prone), nonheterosexuality should be more familial on the maternal than the paternal side. Contrary to this hypothesis, there was no excess of nonheterosexuality among maternal uncles compared to paternal uncles of nonheterosexual males. However, of 320 uncles of nonheterosexual males, only 6 were known to be nonheterosexual. This is half the rate to be expected given the conservative 4% rate in the general population. If homosexuality is familial (between generations), the expected number should be higher, perhaps substantially so. It would appear that subjects have inadequate knowledge about the sexual orientation about their uncles. This is consistent with the comments of several subjects. Subjects presumably did have accurate estimates of whether uncles had ever been married. However, given the far from perfect sensitivity and specificity of this indicator and the low base rate of nonheterosexuality, this is not a very powerful test. Thus, it appears that the examination of nonheterosexuality rates for uncles is not a very good test of the maternal effects hypothesis in the present sample. Nevertheless, the limited and imperfect data available in the present study fail to support the model.

Female nonheterosexuality. The picture is much less clear for female nonheterosexuality than for male nonheterosexuality. This is due,

in part, to the small number of female nonheterosexuals included in the study. This group is, on a priori grounds, the most interesting.

Across the groups, few sisters were known to be nonheterosexual; no significant differences arose when only known nonheterosexuality was considered. When suspected nonheterosexuality was also considered, there was a significant difference in favor of sisters of nonheterosexual females. This finding is suspect, however, as it is not consistent across both the "known" and "suspected" categories. Furthermore, Pillard and Weinrich contacted sisters of nonheterosexual females and found no excess of nonheterosexuality. (See Weinrich, 1987.) Skepticism is also warranted because of the finding of Eckert et al. that four pairs of female MZ twins reared apart were discordant for sexual orientation. No female pairs were concordant for a nonheterosexual orientation.

Suggestions for Future Research

The central finding of the present investigation is the apparent unimportance of the most promising environmental explanation of male homosexuality. The most striking positive result is the replication of a high degree of familiality for male nonheterosexuality. Given this conjunction of findings, it would appear that the most promising line of research on etiology concerns the nature of this familiality. With the exception of attitudes (Loehlin and Nichols, 1976), most familial resemblance heretofore investigated is primarily due to heredity (Plomin and

Daniels, 1987). Whether or not individual differences in sexual orientation are due to genetic differences is an empirical question. Regardless of the answer, the best way of investigating the nature of the familiality is via the methods of population genetics. Possible outcomes for such a research program, along with their interpretations are discussed below.

Environment. Despite the utter failure of previous attempts to explain homosexuality by the environment shared by siblings -- usually, the characteristics of parents -- the importance of common or shared environment has not been disproved. Perhaps investigators have focused on the wrong aspects of shared environment.

The crucial datum in determining the importance of the common environment has traditionally been the correlation between unrelated individuals reared together, or slightly more problematically, the correlation between individuals and their adoptive parents. The latter analysis would not be especially informative or practical for homosexuality. One would not expect adoptive parents to be homosexual (with current adoption practices) regardless of the orientation of their adoptive children. However, the resemblance in sexual orientation of unrelated children reared together would be informative on the importance of common environment.

Actually, such an analysis would be informative only about a certain aspect of common environment -- namely, the common postnatal environment. The prenatal environment shared by (natural, nontwin) siblings may also be important for a trait. This possibility was examined above, e.g., in the discussion of maternal stress-proneness. The

ideal test of this possibility would be to compare rates of nonhetero-sexuality in maternal and paternal half-siblings of nonheterosexuals.

Unshared environment -- those aspects of the environment not shared by siblings reared together -- is more important than shared environment for most behavioral traits (Plomin and Daniels, 1987). The usual indicator of the magnitude of unshared environment has been 1 - r_{MZ}, where r_{MZ} is the correlation of monozygotic (MZ) twins for a trait. However, this may underestimate the importance of unshared environment if prenatal environmental factors are important, since MZ twins share many aspects of the prenatal environment. The main hypothesis of this study involved the unshared prenatal environment -- maternal stress experienced during pregnancy. One test of the importance of the unshared prenatal environment would involve the comparison of male dizygotic (DZ) twin (proband-wise) concordance rates with familiality rates for nontwin siblings. If the unshared prenatal environment is important, then DZ twins should have higher concordance rates for sexual orientation than predicted by the familiality rates for siblings.

Heredity. The evolutionary difficulties faced by genetic explanations of homosexuality need to be taken seriously; however, they do not doom all such attempts to failure. Two basic research strategies can be pursued within a hereditarian framework: direct and indirect strategies. The direct strategy consists of applying the methodologies of population genetics and investigating whether patterns of

resemblance among relatives conform to a genetic hypothesis. For instance, all genetic models specify that MZ twins should resemble each other most closely, followed by DZ twins and nontwin siblings, followed by unrelated individuals reared together. Mixed models, including both genetic and environmental determinants, are possible and indeed likely. In this case, the primary outputs of the direct program will be estimates of the relative importance of various components of variance, including shared and unshared environment, additive and nonadditive genetic effects, and gene-environment interaction and covariance.

Besides the usual predictions made by genetic models, an additional prediction (and a corollary) stems from the apparent low rates of reproductivity among homosexuals. Genes for homosexuality should be disproportionately recessive; therefore, genetic variance should be disproportionately nonadditive. The main prediction stems from the mathematical fact that recessive genes are harder for natural selection to "see," and can be maintained at higher rates than dominant genes despite being evolutionarily maladaptive. The corollary follows from the definition of nonadditive variance (Falconer, 1967). One prediction which follows is that the MZ concordance rate for nonheterosexuality should be significantly greater than twice the DZ concordance rate. This follows from the fact that MZ twins share all nonadditive genetic variance, while DZ twins share less than one-fourth the nonadditive variance. A second prediction is that nonheterosexual males who reproduce should have lower rates of nonheterosexual sons than they have nonheterosexual brothers. This is because parents and offspring do

not share any nonadditive genetic variance.

The indirect hereditarian research program takes as its raison d'etre the need to explain the persistence of the homosexual phenotype in the face of natural selection. If genetic differences are held to underlie differences in sexual orientation, the following must be true: Though genes for homosexuality hurt the homosexual (evolutionarily speaking), they benefit someone else, usually a close relative. The two most commonly discussed relevant models are heterozygote superiority and kin selection.

The classic example of heterozygote superiority is sickle cell anemia. Individuals who are homozygous for the sickling gene become ill and usually die in childhood -- thus, fertility in homozygotes is markedly reduced. Homozygotes -- individuals who have only one sickling gene -- have increased protection from malaria, and thus, increased fertility compared to individuals with no sickling genes (hemizygotes). This advantage of homozygosity is enough to guarantee the persistence of the sickling gene in the population, if at low rates. In order to investigate this possibility for human male homosexuality, one would pursue two strategies: First, one would attempt to establish increased fertility among individuals heterozygous for "gay genes." The predicted difference in fertility is not large compared to effect sizes typically investigated in the behavioral sciences. Assume that homosexuality is caused by being homozygous for a recessive "gay gene." (Assume this for males only; females are unaffected.) Assume further that the prevalence of homosexuality is 3% (as in Fay et al., 1989), and the reduction in fer-

tility of homosexuals is 80%. If the frequency of the gay gene is stable, the ratio of offspring of heterozygotes to homozygotes (i.e., individuals homozygous for the allele of the "gay gene") must be about 1.1. Heterozygotes cannot be identified directly, but should be disproportionately found among parents and heterosexual siblings of homosexual males. In this case, however, the predicted ratio of fertilities shrinks even further due to the imperfect identification of heterozygotes.

A partial test from the indirect program was possible in the present study. Male nonheterosexuals had significantly more siblings, on average, than did the other groups (2.37 versus 1.78, respectively). However, when results were statistically adjusted for mothers' age and parents' education, they no longer differed significantly.

A second method of investigating the heterozygote superiority model of male homosexuality would be to search for qualities of heterozygotes which might conceivably affect their reproductive success. Examples might include attractiveness, intelligence, or characteristics of their reproductive systems -- e.g., the miscarriage rate among females. This latter variable was, in fact, assessed in mothers in the present study; no significant difference was found between the mothers of heterosexuals and noheterosexual males.

Kin selection is identical to heterozygote superiority in its effect. The difference is that kin selection implies that the homosexual makes a behavioral contribution towards his kin, enhancing their fertility at his own expense. It has been suggested, for instance, that homosexuals may help their families in raising siblings and siblings' offspring (Ruse,

1988). Weinrich (1976) studied homosexuality among American Indian tribes. He found that homosexuals were frequently accorded high status by the tribe, and hence were in a position to help their close relatives.

Some sociobiologists (e.g., Ruse, 1988) have speculated that some circumstances, such as childhood illness (or susceptibility to illness), may make homosexuality an individual's best reproductive strategy. That is, certain factors may lower an individual's expected reproductive fitness, hence rendering indirect reproductive strategies more attractive. This hypothesis was behind the inclusion of the item concerning subjects' childhood health in the maternal rating scales. Appreciable differences -- though significant only for males -- were found for both males and females, with nonheterosexuals rated as having been less healthy. Furthermore, for both sexes, the nonheterosexuals had significantly greater variance on the item. This suggests the possibility that childhood illness might appreciably affect the orientation of a few nonheterosexual subjects. Consistent with this possibility, in his study of effeminate boys Green (1987) relates that two of his effeminate subjects were quite ill as children. Unfortunately, he does not give the rate of illness among his comparison group. Though gaining some intriguing support, the hypothesis that homosexuality may be an alternative reproductive strategy when expected fitness is diminished suffers from the lack of specificity of how homosexuality is a reproductive strategy at all.

Some advocates of kin selection (e.g., Weinrich, 1976) is have suggested that the alleged altruistic behaviors of homosexuals probably

made more of a difference in harsher times. If this is true, two facts follow: First, it will be difficult to make a convincing claim that a certain behavior allowed the persistence of the gay gene, since the inclusive fitness of contemporary homosexuals will be diminished. Second, because of this diminished inclusive fitness, the gay gene will become increasingly uncommon, albeit at a slow rate.

Conclusions

The progression of knowledge about homosexuality has differed from that about intelligence or schizophrenia. Regarding the latter traits, we know relatively much about the causes of relevant neurophysiological differences -- their transmission, the relative importance of environment and heredity, their vehicles -- but relatively little about the neurophysiology differences, themselves. The opposite is true of homosexuality. Although the degree of current support for the neuroendocrinological theory is debatable, there are no strong competing theories. Moreover, the theory is reasonably well-specified. However, nothing is known, for instance, about the relative importance of heredity and environment for homosexuality.

This investigation included a test of the most promising vehicle, maternal stress. The results of that test suggest that maternal stress is not a common vehicle for the neurohormonal feminization which

causes male homosexuality. The failure of the maternal stress hypothesis is dissappointing, but barely injurious to the advocate of the neurohormonal theory. Moreover, there is another good lead in the search for a vehicle -- familiality. The advancement of science depends on both the success and failure of theories. Rather than dwell on the failure of the maternal stress hypothesis, let us seek new successes in explaining the fascinating paradox of familial homosexuality.

Appendix 1

Part 1

Questionnaire Completed by Subjects

PARENTAL INTERVIEW RELEASE FORM

We are studying the effects of prenatal stress on aspects of sexuality, such as sexual orientation. In order to do so, it is necessary that we gather information regarding prenatal events from mothers of subjects. This requires that we ask both for your permission to contact your mother and for the address where she can be reached. Before you decide whether to allow us to contact your mother, please note the following:

1. Your mother will have no idea that we are studying aspects of sexual orientation. There will be nothing in the packet we send to indicate this. All she will know is that we are studying the effects of prenatal stress. Please examine a sample packet in order to assure yourself about the subtletly of the questions.

2. Your mother will have no access to any of your responses to our questions. Nor will you have any access to any of her responses.

If you have any questions about this, please consult the experimenter. Your permission is crucial to our experiment. If you decide not to give your permission, please notify the experimenter immediately. Your participation will be no longer required, and you will be given credit for the time you have spent so far. There is no penalty for refusal of permission to contact your mother. However, we hope that you will accept our assurances that there is no need to be concerned, and that you will agree to give permission.

If you agree to let us contact your mother, please sign below:

Signature:_____

If you have agreed, please provide the following information regarding your mother:

Name: Ms./Mrs./Miss_____

Street Address:_____

City/State/Zip:_____

For purposes of comparison, we will also ask your mother about events which occurred during her pregnancy with one of your siblings. Therefore, please provide us with the first name of the heterosexual sibling (if you have one) who is closest in age to you:

Name:_____ Age:_____ Sex:_____

We will need your first name in order to ask your mother about events during her pregnancy with you. Please print neatly your first name, only, below.

Your first name:_____

Please circle the number of the choice which best describes you.

1. I have never had a sexual fantasy about other members of my own sex. My sexual fantasies always involve members of the opposite sex.

2. I have had very few sexual fantasies about other members of my own sex. The vast majority of my sexual fantasies have been about members of the opposite sex.

3. I have had many sexual fantasies about other members of my own sex. However, I more often fantasize about members of the opposite sex.

4. My sexual fantasies are equally often about men and women.

5. I have had many sexual fantasies about members of the opposite sex. However, I more often fantasize about other members of my own sex.

6. I have had very few sexual fantasies about members of the opposite sex. The vast majority of my sexual fantasies have been about other members of my own sex.

7. I have never had a sexual fantasy about members of the opposite sex. My sexual fantasies always involve other members of my own sex.

Please circle the number of the choice which best describes you.

1. I have never had any sexual experiences with another person. (By sexual experience we mean an activity which led to orgasm.)

2. I have never had sex with other members of my own sex. My sexual experiences have always involved members of the opposite sex.

3. I have had very few sexual experiences with other members of my own sex. The vast majority of my sexual experiences have been with members of the opposite sex.

4. I have had several sexual experiences with other members of my own sex. However, most of my sexual experiences have been with members of the opposite sex.

5. My sexual experiences have equally often involved men and women.

6. I have had several sexual experiences with members of the opposite sex. However, most of my sexual experiences have been with other members of my own sex.

7. I have had very few sexual experiences with members of the opposite sex. The vast majority of my sexual experiences have been with other members of my own sex.

8. I have never had a sexual experience with members of the opposite sex. My sexual experiences have always involved other members of my own sex.

BEHAVIOR QUESTIONNAIRE--FEMALES ONLY

(Males please turn over the page.)

Please answer the following questions as they pertained to you during childhood (age 12 and below). Please circle your response:

1. Were you regarded as a tomboy?

a. Yes b. No c. Don't know

2. Were you usually a loner?

a. Yes b. No c. Don't know

3. Did you ever wish you had been a boy rather than a girl?

a. Yes b. No c. Don't know

4. Did you prefer playing or associating with boys rather than girls?

a. Yes b. No c. Don't know

5. Did you prefer dressing in male clothes?

a. Yes b. No c. Don't know

Please answer the following questions as they pertained to you during adolescence (age 13 to age 17). Please circle your response:

1. Were you regarded as a tomboy?

a. Yes b. No c. Don't know

2. Were you usually a loner?

a. Yes b. No c. Don't know

3. Did you ever wish you had been a boy rather than a girl?

a. Yes b. No c. Don't know

4. Did you prefer playing or associating with boys rather than girls?

a. Yes b. No c. Don't know

5. Did you prefer dressing in male clothes?

a. Yes b. No c. Don't know

BEHAVIOR QUESTIONNAIRE--MALES ONLY

(Females please turn over the page.)

Please answer the following questions as they pertained to you during childhood (age 12 and below). Please circle your response:

1. Were you regarded as a sissy?

a. Yes b. No c. Don't know

2. Were you usually a loner?

a. Yes b. No c. Don't know

3. Did you ever wish you had been a girl rather than a boy?

a. Yes b. No c. Don't know

4. Did you prefer playing or associating with girls rather than boys?

a. Yes b. No c. Don't know

5. Did you ever dress up in female clothes (drag)?

a. Yes b. No c. Don't know

Please answer the following questions as they pertained to you during adolescence (age 13 to age 17). Please circle your response:

1. Were you regarded as a sissy?

a. Yes b. No c. Don't know

2. Were you usually a loner?

a. Yes b. No c. Don't know

3. Did you ever wish you had been a girl rather than a boy?

a. Yes b. No c. Don't know

4. Did you prefer playing or associating with girls rather than boys?

a. Yes b. No c. Don't know

5. Did you ever dress up in female clothes (drag)?

a. Yes b. No c. Don't know

RELATIVES QUESTIONNAIRE

Please answer the following questions about your relatives. We are asking about only those brothers, sisters, uncles, and other relatives who are blood relatives. In the questions below, we use the term "gay men" to indicate those men who prefer other men over women as sex partners. "Lesbians" refers to women who prefer other women over men as sex partners. Bisexual men and women are about equally sexually attracted to both sexes. Heterosexual men and women prefer members of the opposite sex as sex partners.

1. How many brothers do you have?_____

2. Of your brothers, how many are older than you?_____

3. How many sisters do you have?_____

4. Of your sisters, how many are older than you?_____

5. Of your brothers, how many are you certain are gay?_____

How many are you certain are bisexual? _____

6. Of your remaining brothers, not counting those referred to in question 5, how many do you suspect might be gay? _____

How many do you suspect may be bisexual? _____

7. How many brothers does your mother have? _____

8. Of these, how many have never married?_____

9. How many of your mother's brothers are you certain are gay?_____

10. How many of your mother's brothers are you certain are bisexual?_____

11. How many of your mother's brothers do you suspect may be gay?_____

12. How many of your mother's brothers do you suspect may be bisexual?_____

13. How many brothers does your father have?_____

14. Of these, how many have never married?_____

15. How many of your father's brothers are you certain are gay?_____

16. How many of your father's brothers are you certain are bisexual?_____

17. How many of your father's brothers do you suspect may be gay?_____

18. How many of your father's brothers do you suspect may be bisexual?_____

19. How many first cousins do you have on your mother's side? _____

20. How many first cousins do you have on your father's side? _____

21. Of your sisters, how many are you certain are lesbian?_____

(Please turn over the page)

How many are you certain are bisexuals? _____

22. Of your remaining sisters, not counting those referred to in question 21, how many do you suspect might be lesbians? _____

How many do you suspect may be bisexual? _____

23. How many sisters does your mother have? _____

24. Of these, how many have never married?_____

25. How many of your mother's sisters are you certain are lesbians?_____

26. How many of your mother's sisters are you certain are bisexual?_____

27. How many of your mother's sisters do you suspect may be lesbians?_____

28. How many of your mother's sisters do you suspect may be bisexual?_____

29. How many sisters does your father have?_____

30. Of these, how many have never married?_____

31. How many of your father's sisters are you certain are lesbians?_____

32. How many of your father's sisters are you certain are bisexual?_____

33. How many of your father's sisters do you suspect may be lesbians?_____

34. How many of your father's sisters do you suspect may be bisexual?_____

35. Have you ever directly revealed your sexual orientation (whether you consider yourself heterosexual, gay or lesbian) to your mother? (Circle)

Yes No Not sure

36. Does your mother know your sexual orientation for certain? Yes No Not sure

37. Does your mother suspect that your sexual orientation is not heterosexual? Yes No Not sure

38. What is your sex? (Male or Female)_____

39. What is your age?_____

40. How many nieces do you have?_____

41. How many nephews do you have?_____

Appendix 1

Part 2

Questionnaire Completed by Subjects

DEPARTMENT OF PSYCHOLOGY
THE UNIVERSITY OF TEXAS AT AUSTIN

Mezes Hall 330 · Austin, Texas 78712

Dear ,

Your child, , has participated in a research project at the University of Texas Department of Psychology, where we are studying the effects of prenatal stress on subsequent personality development. Your child has given us permission to contact you. We hope that you will participate in our study. Your participation involves the following tasks:

1. Filling out the BACKGROUND INFORMATION SHEET.

2. Filling out a rating scale regarding the childhood personality of your child.

3. Filling out the LIFE EVENTS QUESTIONNAIRE, as described in the instructions. This questionnaire involves your memories of stressful life events during your pregnancy with your child, and possibly, during another pregnancy as well.

4. Filling out the PERSONAL VIEWS SURVEY.

The questions should take about 20 minutes. Some of these tasks--particularly the LIFE EVENTS QUESTIONNAIRE--may require you to give intimate information. We have made every effort to insure that this information will be kept completely confidential. The information which you return to us (on the forms and questionnaires which you answer) does not have your last name on it. When the data are entered into the computer, you will be assigned a number, and even your child's name will be erased. Your child will have no access to the information you provide.

If you elect to help us in our study, please fill out the enclosed questionnaires and then return them in the enclosed self-addressed, stamped envelope. Although your participation is crucial to the success of our study, there is no consequence or penalty for your child if you decline. We hope that you will decide to participate, and that the results of this study will add to scientific knowledge.

Sincerely yours,

Lee Willerman, Ph. D.
Professor of Psychology

J. Michael Bailey
Doctoral Candidate

BACKGROUND INFORMATION QUESTIONNAIRE

The children which you will be asked about later are:

(1)_____ and (2)_____

1. Please circle the highest educational level which you have attained:
   a. elementary or junior high school
   b. some high school
   c. graduated high school
   d. some college
   e. graduated college
   f. some graduate work
   g. graduate degree

2. Please circle the educational level of the natural father of the children listed above:
   a. elementary or junior high school
   b. some high school
   c. graduated high school
   d. some college
   e. graduated college
   f. some graduate work
   g. graduate degree

3. How old were you when the first child listed above (1) was born?

   _____

4. How old were you when the second child listed above (2) was born? (Leave blank if only one child is listed.)

   _____

5. Have you spoken to the first child listed above (1) about filling out the questionnaires for this study? (Circle)

   Yes No

Please rate your child, _____, on the following personality traits. We are interested in these personality traits as they were during childhood (age 12 and below), and not how they are now. Please circle the most accurate response.

1.	0 Very Extraverted	1	2	3	4	5 Very Introverted
2.	0 Very Aggressive	1	2	3	4	5 Very Passive
3.	0 Very High activity level	1	2	3	4	5 Very Low activity level
4.	0 Very Athletic	1	2	3	4	5 Very Nonathletic
5.	0 Very Dominant	1	2	3	4	5 Very Submissive
6.	0 Very Emotional	1	2	3	4	5 Very Calm
7.	0 Very Conforming	1	2	3	4	5 Very Nonconforming
8.	0 Very Rigid	1	2	3	4	5 Very Adaptable
9.	0 Very Feminine	1	2	3	4	5 Very Masculine
10.	0 Very Well-adjusted	1	2	3	4	5 Very Poorly adjusted
11.	0 Very Polite	1	2	3	4	5 Very Rude
12.	0 Very Sickly	1	2	3	4	5 Very Healthy

(Please turn over the page.)

LIFE EVENTS QUESTIONNAIRE--INSTRUCTIONS

On the following page is a list of events which could cause emotional stress. We are interested in whether any of these events occurred to you during the period of your life just before and during the pregnancy of the child (or children) named on the enclosed questionnaire(s).

We have broken this period into four smaller periods: the year before conception, and the first, the second, and the third trimesters of pregnancy. For each possible event listed, we would like you to indicate whether it occurred during each of these periods. Please mark a "0" (zero) if the event did not occur. Otherwise, mark a "1", "2", "3", or "4". A "4" should indicate that the event did occur, and that it was extremely stressful. A "3" should indicate that the event did occur, and that it was moderately stressful. A "2" should indicate that the event did occur, and that it was mildly stressful. A "1" should indicate that the event did occur, but that it was not stressful at all. For each event, you should make four marks: one for the year before conception, one for the first trimester of pregnancy, one for the second trimester of pregnancy, and one for the third trimester of pregnancy. If you believe that an event occurred during the pregnancy but cannot remember which trimester, then leave the space for the trimesters in question blank, and make a mark in the last column. The last column should otherwise be left blank.

One last point: We are concerned about the enduring effects of prenatal stress. Therefore, if an event occurred in one period, but continued to cause stress in another period, mark a 1, 2, 3 or 4 for both periods. For example, if a severely distressing event occurred during the first trimester, but caused equally severe stress in the second as well, mark a "4" in the spaces provided for both the first and second trimesters. If it extended into the third trimester, but was less severe, mark a 3 or a 2 in that trimester as well.

Examples

Suppose that you had been a victim of a crime during the year before getting pregnant (and only at this time). If this had caused moderate stress, you would respond as follows:

	Year Before	First Trimester	Second Trimester	Third Trimester	Sometime During Pregnancy
Victim of crime	3	0	0	0	___

If, on the other hand, the stress was moderate when it occurred, and persisted as mild into the first trimester, you would respond as follows:

	Year Before	First Trimester	Second Trimester	Third Trimester	Sometime During Pregnancy
Victim of crime	3	2	0	0	___

If you were certain that you were a victim of crime during pregnancy (causing moderate stress), but could not remember whether this occurred during the second or third trimester, you would respond as follows:

	Year Before	First Trimester	Second Trimester	Third Trimester	Sometime During Pregnancy
Victim of crime	0	0	___	___	3

Note that there is a "0" in the space for the first trimester. This is because the crime may have occurred during either the second or third, but not the first trimester.

ID:

LIFE EVENTS QUESTIONNAIRE (continued)

Please indicate which of the following life events you experienced in the year before and in the months during your pregnancy with _____.

Rate the severity of stress as described in the instructions. ("4" indicates severe stress, "3" indicates moderate stress, "2" indicates mild stress, and "1" indicates that the event occurred but caused no stress. "0" indicates that the event did not occur during the given period.)

Event	Year Before	First Trimester	Second Trimester	Third Trimester	Sometime During Pregnancy
1. Victim of a crime	____	____	____	____	____
2. Legal troubles	____	____	____	____	____
3. Change in pattern of schooling	____	____	____	____	____
4. Problems in school	____	____	____	____	____
5. Change in pattern of work or job.	____	____	____	____	____
6. Problems in job	____	____	____	____	____
7. Moved your residence	____	____	____	____	____
8. Pregnancy was unplanned	____	____	____	____	____
9. Pregnancy was unwanted	____	____	____	____	____
10. Pregnancy was difficult	____	____	____	____	____
11. Marital problems	____	____	____	____	____
12. Problems with other family members or friends	____	____	____	____	____
13. Spouse was away from you for an extended time	____	____	____	____	____
14. Death of friend	____	____	____	____	____
15. Death of family member	____	____	____	____	____
16. Financial problems	____	____	____	____	____
17. You had a physical illness or injury	____	____	____	____	____
18. Family member had a physical illness or injury	____	____	____	____	____

(see other side)

LIFE EVENTS QUESTIONNAIRE (continued)

	Year Before	First Trimester	Second Trimester	Third Trimester	Sometime During Pregnancy
19. You had a mental illness	____	____	____	____	____
20. Family member had a mental illness	____	____	____	____	____
21. Vaginal bleeding	____	____	____	____	____
22. Unmarried status	____	____	____	____	____
23. Involved in automobile accident	____	____	____	____	____
24. Saw a counselor of some kind	____	____	____	____	____
25. Job loss to you or spouse	____	____	____	____	____

The next 3 items inquire about your use of certain substances. Mark a "0" if you did not use the substance at all during the specified period, a "1" if you used it once, a "2" if you used it several times, a "3" if you used it often, and a "4" if you used it every day.

26. Used barbiturates	____	____	____	____	____
27. Used alcohol	____	____	____	____	____
28. Used other drugs (please specify)	____	____	____	____	____

The following spaces are provided for you to fill in information about any stressful events which we have not covered. Please write a brief description of the event and rate its stressfulness.

29.	____	____	____	____	____
30.	____	____	____	____	____
31.	____	____	____	____	____
32.	____	____	____	____	____

33. Have you ever miscarried (lost a child during pregnancy)? (Circle) Yes No

PERSONAL VIEWS SURVEY

Below are some items that you may agree or disagree with. Please indicate how you feel about each one by circling a number from 0 to 3 in the space provided. A zero indicates that you feel the item is not at all true; circling a three means that you feel the item is completely true.

As you will see, many of the items are worded very strongly. This is to help you decide the extent to which you agree or disagree.

Please read all the items carefully. Be sure to answer all on the basis of the way you feel now. Don't spend too much time on any one item.

0 = Not at all true
1 = A little true
2 = Quite a bit true
3 = Completely true

1. I often wake up eager to take up my life where it left off the day before.....	0	1	2	3
2. I like a lot of variety in my work.....	0	1	2	3
3. Planning ahead can help avoid most future problems.....	0	1	2	3
4. I usually feel that I can change what might happen tomorrow, by what I do today.....	0	1	2	3
5. I feel uncomfortable if I have to make any changes in my everyday schedule.....	0	1	2	3
6. No matter how hard I try, my efforts will accomplish nothing.....	0	1	2	3
7. I find it difficult to imagine getting excited about working.....	0	1	2	3
8. No matter what you do, the "tried and true" ways are always the best.....	0	1	2	3
9. I feel that it's almost impossible to change my spouse's mind about something.....	0	1	2	3
10. New laws shouldn't be made if they hurt a person's income.....	0	1	2	3
11. When you marry and have children you have lost your freedom of choice.....	0	1	2	3
12. No matter how hard you work, you never really seem to reach your goals.....	0	1	2	3
13. A person whose mind seldom changes can usually be depended on to have reliable judgment.....	0	1	2	3
14. I believe most of what happens in life is just meant to happen.....	0	1	2	3
15. I don't like conversations when others are confused about what they mean to say.....	0	1	2	3
16. Most of the time it just doesn't pay to try hard, since things never turn out right anyway.....	0	1	2	3
17. The most exciting thing for me is my own fantasies.....	0	1	2	3
18. I won't answer a person's questions until I am very clear as to what he is asking.....	0	1	2	3
19. When I make plans I'm certain I can make them work.....	0	1	2	3
20. I really look forward to my work.....	0	1	2	3
21. It doesn't bother me to step aside for a while from something I'm involved in, if I'm asked to do something else.....	0	1	2	3
22. It's exciting for me to learn some thing about myself.....	0	1	2	3
23. I enjoy being with people who are unpredictable.....	0	1	2	3
24. I find it's usually very hard to change a friend's mind about something.....	0	1	2	3

(Please turn over the page.)

0 = Not at all true
1 = A little true
2 = Quite a bit true
3 = Completely true

25. Thinking of yourself as a free person just makes you feel frustrated and unhappy.....	0	1	2	3
26. It bothers me when something unexpected interrupts my daily routine.....	0	1	2	3
27. When I make a mistake, there's very little I can do to make things right again.....	0	1	2	3
28. I feel no need to try my best at work, since it makes no difference anyway.....	0	1	2	3
29. I respect rules because they guide me.....	0	1	2	3
30. One of the best ways to handle most problems is just not to think about them.....	0	1	2	3
31. I believe that most athletes are just born good at sports.....	0	1	2	3
32. I don't like things to be uncertain or unpredictable.....	0	1	2	3
33. People who do their best should get full financial support from society.....	0	1	2	3
34. Most of my life gets wasted doing things that don't mean anything.....	0	1	2	3
35. Lots of times I don't really know my own mind.....	0	1	2	3
36. I have no use for theories that are not closely tied to the facts.....	0	1	2	3
37. Ordinary work is just too boring to be worth doing.....	0	1	2	3
38. When other people get angry at me, it's usually for no good reason.....	0	1	2	3
39. Changes in routine bother me.....	0	1	2	3
40. I find it hard to believe people who tell me that the work they do is of value to society.....	0	1	2	3
41. I feel that if someone tries to hurt me, there's usually not much I can do to try and stop him.....	0	1	2	3
42. Most days, life just isn't very exciting for me.....	0	1	2	3
43. I think people believe in individuality only to impress others.....	0	1	2	3
44. I want to be sure someone will take care of me when I get old.....	0	1	2	3
45. Politicians run our lives.....	0	1	2	3

Personal Views Survey: Part II

Please rate each of the items on a scale of 1 (not characteristic or typical of yourself) to 5 (very characteristic or typical of yourself.)

_____ 1. I like to be with people.

_____ 2. I usually seem to be in a hurry.

_____ 3. I am easily frightened.

_____ 4. I frequently get distressed.

_____ 5. When displeased, I let people know it right away.

_____ 6. I am something of a loner.

_____ 7. I like to keep busy all the time.

_____ 8. I am known as hotblooded and quick-tempered.

_____ 9. I often feel frustrated.

_____ 10. My life is fast paced.

_____ 11. Everyday events make me troubled and fretful.

_____ 12. I often feel insecure.

_____ 13. There are many things that annoy me.

_____ 14. When I get scared, I panic.

_____ 15. I prefer working with others rather than alone.

_____ 16. I get emotionally upset easily.

_____ 17. I often feel as if I'm bursting with energy.

_____ 18. It takes a lot to make me mad.

_____ 19. I have fewer fears than most people my age.

_____ 20. I find people more stimulating than anything else.

APPENDIX 2
Intercorrelations among Childhood Gender Nonconformity Items

(MALES)	S1	S2	S3	S4	S5	M1	M2	M3	M4	M5
Self-rated items										
S1 Sissy	1.00									
S2 Loner	0.27	1.00								
S3 Wanted to be female	0.36	0.07	1.00							
S4 Pref. female company	0.49	0.27	0.43	1.00						
S5 Pref. female dress	0.19	0.00	0.37	0.18	1.00					
Maternally-rated items										
M1 Introverted	0.10	0.11	0.05	0.10	-0.01	1.00				
M2 Passive	0.03	0.03	-0.12	-0.00	-0.02	0.50	1.00			
M3 Low activity	0.18	0.03	0.04	0.16	0.05	0.50	0.36	1.00		
M4 Nonathletic	0.35	0.25	0.26	0.31	0.15	0.31	0.20	0.40	1.00	
M5 Submissive	0.09	-0.06	0.04	0.12	0.17	0.37	0.48	0.34	0.35	1.00
M6 Calm	-0.12	0.08	0.00	-0.08	-0.04	0.15	0.20	0.14	0.18	0.22
M7 Nonconforming	0.13	0.08	0.02	0.04	-0.10	-0.05	-0.07	0.06	0.06	-0.24
M8 Adaptable	-0.10	-0.18	-0.10	-0.14	0.10	-0.22	-0.03	0.03	0.06	0.17
M9 Masculine	-0.32	-0.15	-0.16	-0.22	-0.06	-0.22	-0.16	-0.26	-0.39	-0.08
M10 Poorly adjusted	0.15	0.18	0.08	0.14	0.02	0.20	0.13	0.19	0.21	0.15
M11 Rude	0.11	0.13	0.09	0.10	-0.02	-0.02	-0.07	0.17	0.09	-0.02
M12 Healthy	-0.37	-0.12	-0.10	-0.22	-0.02	-0.08	-0.11	-0.08	-0.08	0.10
Scales										
SS Self-rated scale	0.72	0.54	0.66	0.74	0.54	0.11	-0.01	0.14	0.40	0.12
MS Maternal scale	0.30	0.14	0.12	0.27	0.11	0.71	0.68	0.71	0.72	0.65

APPENDIX 2 (cont'd.)

(MALES)	M6	M7	M8	M9	M10	M11	M12	SS	MS
Maternally-rated items									
M6 Calm	1.00								
M7 Nonconforming	-0.17	1.00							
M8 Adaptable	0.16	-0.13	1.00						
M9 Masculine	0.08	-0.13	0.19	1.00					
M10 Poorly adjusted	-0.19	0.19	-0.21	-0.32	1.00				
M11 Rude	-0.12	0.38	-0.06	-0.04	0.52	1.00			
M12 Healthy	0.16	-0.19	0.25	0.25	-0.09	-0.00	1.00		
Scales									
SS Self-rated scale	-0.04	0.05	-0.14	-0.28	0.17	0.11	-0.26	1.00	
MS Maternal Scale	0.19	-0.01	-0.03	-0.50	0.30	0.07	-0.14	0.29	1.00

All correlations involving maternally-rated items and scales are based on a minimum of 141 subjects. Other correlations are based on a minimum of 199 subjects. All correlations ≥ 0.17 in absolute value are significant.

APPENDIX 2 (cont'd.)

(FEMALES)	S1	S2	S3	S4	S5	M1	M2	M3	M4	M5
Self-rated items										
S1 Tomboy	1.00									
S2 Loner	0.28	1.00								
S3 Wanted to be male	0.46	0.30	1.00							
S4 Pref. male company	0.31	0.22	0.24	1.00						
S5 Pref. male dress	0.60	0.17	0.46	0.28	1.00					
Maternally-rated items										
M1 Introverted	-0.21	-0.13	-0.12	-0.22	-0.14	1.00				
M2 Passive	-0.18	-0.16	-0.25	-0.36	-0.17	0.64	1.00			
M3 Low activity	-0.28	0.01	-0.23	-0.14	-0.18	0.39	0.51	1.00		
M4 Nonathletic	-0.30	0.09	-0.11	-0.42	-0.16	0.21	0.37	0.59	1.00	
M5 Submissive	-0.15	-0.21	-0.27	-0.31	-0.20	0.31	0.54	0.22	0.16	1.00
M6 Calm	-0.11	-0.03	-0.25	-0.02	-0.07	0.04	0.13	0.25	0.11	0.43
M7 Nonconforming	0.09	0.14	0.24	0.15	0.10	-0.03	-0.22	0.17	0.16	-0.49
M8 Adaptable	0.02	0.09	-0.03	0.00	0.07	-0.01	0.16	0.11	-0.02	0.31
M9 Masculine	0.47	0.07	0.33	0.08	0.49	-0.08	0.00	0.02	-0.22	-0.02
M10 Poorly adjusted	0.11	0.21	0.22	-0.12	0.13	0.43	0.29	0.32	0.45	0.09
M11 Rude	0.02	-0.19	0.11	-0.16	-0.00	0.23	0.04	0.07	0.26	-0.11
M12 Healthy	-0.01	-0.26	-0.01	-0.05	0.03	0.08	0.21	0.33	0.04	0.09
Scales										
SS Self-rated scale	0.80	0.56	0.72	0.60	0.74	-0.24	-0.32	-0.24	-0.27	-0.33

APPENDIX 2 (cont'd.)

(FEMALES)	M6	M7	M8	M9	M10	M11	M12	SS
Maternally-rated items								
M6 Calm	1.00							
M7 Nonconforming	-0.20	1.00						
M8 Adaptable	0.53	-0.09	1.00					
M9 Masculine	-0.08	0.13	-0.01	1.00				
M10 Poorly adjusted	-0.19	0.19	-0.06	0.08	1.00			
M11 Rude	-0.14	0.24	-0.09	0.08	0.37	1.00		
M12 Healthy	0.08	-0.03	0.03	0.21	-0.03	-0.04	1.00	
Scales								
SS Self-rated scale	-0.14	0.20	0.04	0.42	0.16	-0.06	-0.07	1.00

All correlations involving maternally-rated scales are based on a minimum of 71 subjects. Other correlations are based on 97 subjects. For the smaller N, all correlations ≥ 0.23 in absolute value are significant. For the larger N, all correlations ≥ 0.21 in absolute value are significant.

Appendix 3

Frequency Distributions of Self-Rated Childhood Gender Nonconformity Items

MALES

Item 1: Were you regarded as a sissy?

	Yes		Uncertain		No	
Nonhet. Males	51	(44.0%)	16	(13.8)	49	(42.2)
Het. Males	6	(7.1)	4	(4.7)	75	(88.2)

Item 2: Were you usually a loner?

	Yes		Uncertain		No	
Nonhet. Males	57	(49.6)	0	(0.0)	58	(50.4)
Het. Males	19	(22.4)	2	(2.4)	64	(75.3)

Item 3: Did you wish to be a girl?

	Yes		Uncertain		No	
Nonhet. Males	29	(25.2)	2	(1.7)	84	(73.0)
Het. Males	3	(3.5)	3	(3.5)	79	(92.9)

Item 4: Did you prefer associating with girls?

	Yes		Uncertain		No	
Nonhet. Males	57	(49.1)	6	(5.2)	53	(45.7)
Het. Males	1	(1.2)	9	(10.6)	75	(88.2)

Item 5: Did you prefer dressing in girls' clothing?

	Yes		Uncertain		No	
Nonhet. Males	40	(34.5)	0	(0.0)	76	(65.5)
Het. Males	12	(14.1)	4	(4.7)	69	(81.2)

Appendix 3 (Cntd.)

Frequency Distributions of Self-Rated Childhood Gender Nonconformity Items

FEMALES

Item 1: Were you regarded as a tomboy?

	Yes		Uncertain		No	
Nonhet. Females	17	(68.0%)	2	(8.0)	6	(24.0)
Het. Females	27	(37.5)	5	(6.9)	40	(55.6)

Item 2: Were you usually a loner?

	Yes		Uncertain		No	
Nonhet.	10	(40.0)	2	(8.0)	13	(52.0)
Het. Females	9	(12.5)	0	(0.0)	63	(87.5)

Item 3: Did you wish to be a boy?

	Yes		Uncertain		No	
Nonhet. Females	11	(44.0)	0	(0.0)	14	(56.0)
Het. Females	11	(15.3)	1	(1.4)	60	(83.3)

Item 4: Did you prefer associating with boys?

	Yes		Uncertain		No	
Nonhet. Females	10	(40.0)	0	(0.0)	15	(60.0)
Het. Females	14	(19.4)	4	(5.6)	54	(75.0)

Item 5: Did you prefer dressing in boys' clothing?

	Yes		Uncertain		No	
Nonhet. Females	15	(60.0)	1	(4.0)	9	(36.0)
Het. Females	10	(13.9)	4	(5.6)	58	(80.6)

Appendix 4

Correlations Between Sexual Orientation and Individual Stressors, by Time Period: Males[a]

Stressor	Time			
	Year Before Pregnancy	First Trimester	Second Trimester	Third Trimester
Victim of a crime	0.07	0.01	0.07	0.01
Legal Troubles	0.03	0.06	0.11	0.12
Change in schooling	-0.15	-0.09	0.04	-0.04
Problems in school	-0.21*	0.06	0.07	0.07
Changes in job pattern	-0.11	-0.11	-0.09	-0.13
Problems in job	-0.10	-0.01	0.01	-0.11
Moved residence	-0.01	-0.10	-0.06	-0.08
Unplanned pregnancy	0.17*	-0.05	0.00	0.03
Unwanted pregnancy	0.12	0.03	0.05	0.11
Difficult pregnancy	-0.05	-0.06	-0.09	0.00
Marital problems	0.00	-0.04	0.03	0.01
Problems with family	-0.06	-0.07	-0.09	-0.09
Spouse was away	-0.13	-0.05	0.06	-0.08
Death of friend	0.04	0.12	0.07	0.07
Death of family member	0.02	0.12	-0.10	0.11
Financial problems	0.09	-0.01	-0.02	-0.02
Illness or injury to self	0.05	-0.05	-0.09	0.04

Appendix 4 (ctd.)

Stressor	Time			
	Year Before Pregnancy	First Trimester	Second Trimester	Third Trimester
Illness or injury to family	-0.02	-0.02	-0.02	-0.03
Mental illness: Self	-0.00	-0.03	-0.03	-0.03
Mental illness: Family	-0.05	-0.04	-0.04	-0.02
Vaginal bleeding	-0.12	-0.04	-0.01	-0.07
Unmarried status	0.04	-0.06	-0.05	0.00
Automobile accident	0.01	0.04	0.11	0.05
Saw counselor	-0.00	-0.09	-0.08	-0.08
Job loss to self or spouse	-0.01	-0.07	-0.02	-0.07
Used barbiturates	0.02	-0.03	-0.06	-0.03
Used alcohol	-0.23**	-0.17*	-0.12	-0.13
Used other drugs	0.13	0.12	0.11	0.04

[a] Positive correlations mean the occurrence of an event is associated with a nonheterosexual orientation.

All correlations are based on a minimum of 143 subjects.

*P < .05; **P < .01.

Appendix 4

Correlations Between Sexual Orientation and Individual Stressors, by Time Period: Females[a]

Stressor	Time			
	Year Before Pregnancy	First Trimester	Second Trimester	Third Trimester
Victim of a crime	-0.13	0.00	0.00	0.00
Legal Troubles	-0.02	-0.08	0.03	0.00
Change in schooling	0.11	0.02	0.11	-0.09
Problems in school	0.05	0.00	0.00	0.00
Changes in job pattern	0.05	-0.15	0.16	0.10
Problems in job	0.08	0.19	0.06	0.11
Moved residence	0.22	0.06	0.09	0.18
Unplanned pregnancy	0.22	0.05	0.20	0.19
Unwanted pregnancy	0.26*	0.02	0.17	0.17
Difficult pregnancy	0.10	0.11	0.16	-0.04
Marital problems	0.07	0.11	0.06	0.06
Problems with family	0.11	0.07	0.08	0.10
Spouse was away	-0.02	-0.10	0.13	0.09
Death of friend	0.00	0.00	0.00	0.00
Death of family member	-0.03	-0.13	0.00	0.30**
Financial problems	0.16	0.12	0.21	0.13
Illness or injury to self	0.01	-0.12	-0.14	-0.00
Illness or injury to family	-0.08	-0.10	-0.09	-0.07

Appendix 4 (ctd.)

Stressor	Time			
	Year Before Pregnancy	First Trimester	Second Trimester	Third Trimester
Mental illness: Self	0.00	0.00	0.00	0.00
Mental illness: Family	0.34**	0.29*	0.13	0.13
Vaginal bleeding	-0.03	-0.08	0.19	0.03
Unmarried status	-0.06	-0.08	0.00	0.00
Automobile accident	-0.01	-0.09	0.04	-0.05
Saw counselor	-0.09	-0.09	-0.09	-0.09
Job loss to self or spouse	0.24*	0.34**	0.49***	0.37**
Used barbiturates	-0.09	-0.09	-0.09	-0.09
Used alcohol	-0.20	-0.27*	-0.22	-0.24*
Used other drugs	0.11	0.18	0.17	0.14

[a] Positive correlations mean the occurrence of an event is associated with a nonheterosexual orientation.

All correlations are based on a minimum of 73 subjects.

*P < .05; **P < .01; ***P < .001.

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